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PREVENTIVE MEDICINE AND HYGIENE

PREVENTIVE MEDICINE AND HYGIENE

BY

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WITH CHAPTERS UPON

SEWAGE AND GARBAGE, BY GEORGE C. WHIPPLE, PROFESSOR OF SANITARY ENGINEERING, HARVARD

VITAL STATISTICS, BY CRESSY L. WILBUR, CHIEF STATISTICIAN, BUREAU OF THE CENSUS, DEPARTMENT OF COMMERCE AND LABOR

THE PREVENTION OF MENTAL DISEASES, BY THOMAS W. SALMON, DIRECTOR OF SPECIAL STUDIES, NATIONAL COMMITTEE FOR MENTAL HYGIENE, ETC.

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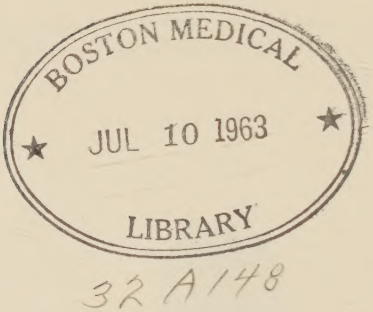

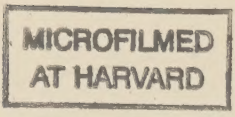

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PREFACE

This book has been written in response to a demand for a treatise based upon modern progress in hygiene and sanitation. The work is planned to include those fields of the medical and related sciences which form the foundation of public health work. So far as I know, no other book on the subject covers the broad field considered in this volume. The progress in hygiene and sanitation has been so rapid that the subject of preventive medicine has become a specialty, and its scope has become so broad that the question throughout the making of this book has been rather what to leave out than what to include. The facts here brought together are widely scattered in the literature and many of them are difficult of access; they have been collected for the convenience of the student of medicine and the physician, as well as those engaged in sanitary engineering or public health work.

During twenty-three years of varied experience in public health work it has been my good fortune to have served as quarantine officer, in epidemic campaigns, in epidemiological investigations, and in public health laboratories, at home, on the Continent, and in the tropics. The fruits of these experiences are reflected in this book, which may be taken as representing my personal views gained in the field, in the laboratory, in the classroom, and in administrative offices.

It is wellnigh impossible to prevent or suppress a communicable disease without a knowledge of its mode of transmission. This is the most important single fact for successful personal prophylaxis, as well as in the general warfare against infection; therefore, the communicable diseases have been grouped in accordance with their modes of transference. Each one of the important communicable diseases is discussed separately in order to bring out the salient points upon which prevention is based. The classification adopted is believed to be unique and should prove helpful to those who are especially concerned in the prevention of infection.

The book may be considered in two parts, namely, that which deals with the person (hygiene) and that which deals with the environment (sanitation). The first part includes the prevention of the communicable diseases, venereal prophylaxis, heredity, immunity, eugenics, and similar subjects. The second part deals with our environment in its relation to health and disease and includes a discussion of food, water, air, soil, disposal of wastes, vital statistics, diseases of occupation, industrial hygiene, school hygiene, disinfection, quarantine, isolation, and other topics of sanitary importance, as well as subjects of interest to health officers. All the important methods used in public health laboratories are described.

To have made this book in monographic style with references to authorities for every statement would have resulted in an unwieldy work of impractical size and form. The textbook style has therefore been adopted and citation of authorities for facts that are now well established has been regarded as unnecessary. In this respect it may seem that I have given scant credit to many workers from whose writings I have borrowed results, thoughts, and sometimes words or even sentences. At the end of each chapter will be found a list of references to articles or books that I have especially drawn upon, and I desire to acknowledge my obligations to these sources as well as to refer the reader to them for further study of particular subjects. I have also drawn freely upon my own previous writings and those of my co-workers in compiling this book. The chapter on "Disinfection" is based upon my book entitled: "Disinfection and Disinfectants," published by P. Blakiston's Sons & Co., Philadelphia, 1902.

I have received generous help from a number of friends and it is a pleasure here to acknowledge especially my obligation to Dr. David L. Edsall for reading and correcting the chapter on "Diseases of Occupation," to Dr. John F. Anderson and Dr. Joseph Goldberger for revising the chapters upon "Measles" and "Typhus Fever," to Prof. George C. Whipple for reading and improving the chapter upon "Water," to Charles T. Brues for many suggestions in the section upon insect-borne diseases, and to Prof. W. E. Castle for a similar service with the section on "Heredity." Dr. Charles Wardell Stiles has kindly furnished information concerning the relation of parasites to soil. I also desire to express my obligations to Prof. Arthur I. Kendall, Dr. Harold L. Amoss,

Dr. Lewis W. Hackett, Prof. William D. Frost, and Miss Emily G. Philpotts.

It has been my object to give in this volume the scientific basis upon which the prevention of disease and the maintenance of health must rest. Exact knowledge has taken the place of fads and fancies in hygiene and sanitation; the capable health officer now possesses facts concerning infections which permit their prevention and even their suppression in some instances. Many of these problems are complicated with economic and social difficulties, which are given due consideration, for preventive medicine has become a basic factor in sociology.

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PREVENTIVE MEDICINE

SECTION I

PREVENTION OF THE COMMUNICABLE DISEASES

CHAPTER I

DISEASES HAVING SPECIFIC OR SPECIAL PROPHYLACTIC MEASURES

SMALLPOX AND VACCINATION

The prevention of smallpox depends primarily upon vaccination, secondarily upon isolation and disinfection. Vaccination was the first specific prophylactic measure given to man; it produces an active immunity to smallpox (variola). On account of its importance and great practical value this subject will be considered in some detail, for much of the antivaccination sentiment is due to ignorance or misconstruction of the facts.

Historical Note.—The credit of giving vaccination to the world is due to Jenner, who proved through carefully planned experiments that cowpox protects against smallpox. This fact had been familiar to the farmers and folk of England as a vague tradition for a long time. A young girl who sought medical advice of Jenner, when a student at Sudbury, said, "I cannot take smallpox because I have had cowpox"; this remark made a strong impression upon the young medical student.

Benjamin Jesty, a Dorchestershire farmer, in 1774 successfully vaccinated his wife and two sons. Plett, in Holstein, in 1791 also successfully vaccinated three children. It was Jenner, however, who through logical and scientific methods proved that a person who has had the mild disease, cowpox, enjoys protection against the serious and often fatal disease, smallpox. Waterhouse and others soon repeated and corroborated Jenner's experiments and helped to establish the soundness of his conclusions.

Jenner made his crucial experiments in 1796, when he transferred the vaccine matter from the hand of a dairy maid (Sarah Nelms) to the arm of a boy about 8 years old—name not given. Sarah Nelms

scratched her hand with a thorn and "was infected with the cowpox from her master's cows, in May, 1796." Jenner transferred the vaccine virus from the eruption upon the hand of Sarah Nelms to the arm of the 8-year-old boy on May 14, 1796. A typical take followed. "In order to ascertain whether the boy, after feeling so slight an affection of the system from the cowpox virus, was secure from the contagion of the smallpox, he was inoculated the first of July following with variolous matter, immediately taken from a pustule. Several slight punctures and incisions were made on both arms, and the matter was carefully inserted, but no disease followed. The same appearances were observable on the arm as we commonly see when a patient has had variolous matter applied, after having either the cowpox or the smallpox. Several months afterward he was again inoculated with variolous matter, but no sensible effect was produced on the constitution."

In addition to such direct experimental proof, Jenner inoculated smallpox matter into ten persons who had at some previous time contracted cowpox.

Date of Inoculation with Smallpox	Name	Ascertained to Have Had Cowpox
1. 1778	Mrs. H. ———	When very young
2. 1791	Mary Barge	31 years previously
3. 1792	Sarah Portlock	27 years previously
4. } 1795	Joseph Merret	25 years previously
5. }	William Smith	1, 5, 15 years previously
6.	Elizabeth Wynne	10 months previously
7. } 1797	Sarah Wynne	9 months previously
8. }	William Rodway	38 years previously
9. After 1782	Simon Nichols	Some years previously
10. Not stated	John Phillips	53 years previously

In justification of such human experimentation it should be remembered that at that time the inoculation of smallpox matter into healthy individuals was an acknowledged method of preventing that disease. Jenner himself was inoculated when a boy. The question of "inoculation" (with smallpox) as contrasted with "vaccination" (with cowpox) will be discussed presently.

With such proof as this Jenner put a popular belief upon a scientific basis. He demonstrated that cowpox is a local and trivial disease in man, that it may be readily transferred from man to man, and that it protects against smallpox. The chain of evidence was complete, but he first proved his thesis to his own satisfaction before he gave it to the world. He said himself: "I placed it on a rock where I knew it would be immovable before I invited the public to take a look at it." Jenner presented the results of his observations to the Royal Society, of which he was a Fellow, but the paper was refused. He then published

it in 1798 as a book, modestly entitled, "An Inquiry Into the Causes and Effects of the Variolæ Vaccinæ, a Disease Discovered in Some of the Western Counties of England, Particularly Gloucestershire, and Known by the Name of the Cowpox." Every student of preventive medicine should read this brief "inquiry" in the original. It may be taken as a model of accurate observation and logical presentation, showing great self-restraint and moderation of an observant, imaginative, and judicial mind.

Dr. Benjamin Waterhouse, the first professor of Theory and Practice of Physic in the Harvard Medical School, early became convinced of the value of Jenner's demonstration and obtained some vaccine virus from abroad. On July 8, 1800, he vaccinated his son, Daniel Oliver Waterhouse, then five years old. This was the first person vaccinated in America, so far as existing records show. Thomas Jefferson helped materially to spread the new doctrine in this country, and, in 1806, in writing to Jenner, said: "Future nations will know by history only that the loathsome smallpox has existed and by you has been extirpated." This prophecy has not yet been fulfilled—though eminently possible.

VACCINATION

Vaccination may be defined as the transference of the virus from the skin eruption of an animal having vaccinia or cowpox into the skin of another animal. For over one hundred years vaccination (from *vacca*—a cow) was a specific term limited to the introduction of the virus of cowpox into the skin, in order to induce vaccinia and prevent variola. In recent years, however, the term has been used in a generic sense to include the introduction of many different substances in many different ways and for many different purposes. Thus we speak of attenuated or killed bacterial cultures as bacterial vaccines; and the subcutaneous inoculation of organic substances of diverse origin and nature is often spoken of as vaccination. We hear of typhoid vaccines, anthrax vaccines, staphylococcus vaccines, and we read in the literature of animals "vaccinated" with extracts of cancer and other organic substances. For distinction between a vaccine and a virus, see page 344.

VACCINE VIRUS

Vaccine virus is the specific principle in the matter obtained from the skin eruption of animals having a disease known as "vaccinia" or "cowpox." Vaccine virus is obtained from calves, man, the buffalo, sometimes the camel, and other animals.

Cowpox, or vaccinia, is an acute specific disease to which many animals are susceptible, namely, man, cattle, camels, rabbits, monkeys,

guinea-pigs, rats, etc. The disease runs practically the same clinical course in all susceptible species. The eruption is always[1] local and confined to the site of the vaccinated area; the constitutional symptoms are always benign and usually slight. Vaccinia or cowpox is a benign disease; when uncomplicated, it has never been known to cause death or leave any unpleasant sequelæ.

After an incubation period of from three to four days the local eruption begins as a papule which soon develops into a vesicle, and later into an umbilicated pustule. Surrounding the vesicle is a reddened, inflamed, and tender areola. The neighboring lymph glands are swollen and tender, and there is slight fever lasting several days. The pustule dries, leaving a crust or scab, which comes away, disclosing a typical foveated or pitted scar.

Human and Bovine Vaccine Virus.—Vaccine virus may be obtained either from bovine or human sources.

Human virus is now seldom used, for the reason that the supply would not be sufficient. Upon the appearance of a smallpox outbreak it is sometimes necessary to have enough virus to vaccinate from one hundred thousand to a million people. Such large quantities evidently could not be obtained from man at any desired time. Another objection to the use of human virus is the possibility, although small, of transmitting syphilis, and perhaps other diseases. When human seed is used the virus may be transferred directly from arm to arm; or the virus may be preserved dry in the scab; or the contents of the vesicle may be kept in either a dried or moist state, as described below for bovine virus. Arm to arm vaccination is still practiced in several parts of the world, particularly in Mexico, where it is claimed that it has the advantage of producing a better take; that the results are surer in that there are fewer unsuccessful vaccinations; and, finally, it is stated that the human virus affords a better immunity, but as to this there is no proof and some doubt.

Bovine virus has been used more or less since the time of Jenner, but especially since Copeman showed in 1891 how to purify it with glycerin. It has the great advantage of being readily obtained in any amount and when desired. It further totally eliminates the danger of conveying syphilis and other diseases peculiar to man.

Forms of Vaccine Virus.—Vaccine virus may be used in one of three forms: (1) fresh, (2) dry, (3) glycerinated.

The fresh virus may be taken from the eruption of the calf or man and transferred directly. Thus the Institut Vaccinale at Paris still prefers to use the fresh virus. The vesicle is squeezed at its base between the blades of forceps, and some of the contents are transferred

[1] Rare exceptions to this statement will be noted later.

directly from the calf to the skin of the arm by means of a thumb lancet or any similar instrument.

The vaccinal matter may be dried, and the virus remains potent in this state a very long time, especially if kept cold and protected from the light. The virus may be dried upon a splinter of ivory or other substance. Formerly physicians preserved the dried crust from a typical take. Small portions of this crust were ground, moistened, and then inserted into the skin.

Glycerinated virus consists of vaccine pulp treated with 60 per cent. pure glycerin. This purifies it and hence is preferable. Before taking up the question of glycerination, we must understand the difference between vaccine lymph and vaccine pulp.

Vaccine Pulp and Vaccine Lymph.—A distinction is drawn between the pulp and the lymph. The *pulp* consists of the entire vesicle with its contents, which is scraped from the skin, and is composed of epithelium, leukocytes, bacteria, products of inflammatory reaction, the fluid content of the vesicle, débris, etc. The *lymph* is the serous fluid contained in the vesicle or which often exudes from the broken vesicle. When the eruption is produced on the skin of a calf in a large confluent area, the surface of the eruption is scraped away and the exuding lymph is placed upon points by dipping or brushing. Most of the active principle of vaccine virus is contained in the epithelial cells, and this portion is largely lost when only the lymph is used. The pulp, which includes the lymph, therefore contains the virus in greater concentration, and is almost exclusively used in this country at the present time.

Dry Points Versus Glycerinated Vaccine Virus.—The old-fashioned dry points were prepared by dipping splinters of ivory into the vaccine lymph. Later the lymph was collected upon a brush and thus transferred to the ivory point. Bone or glass may be substituted for ivory. Bone is undesirable because it is exceedingly difficult to sterilize. The only advantage of the dry point is its convenience in vaccinating. Its disadvantage is that the virus dried upon such points cannot be purified as is the case with glycerinated pulp. Further, the points are used as scarifiers and the method of scarification favors irritation and infection of the wound. The dry points practically always contain more bacteria than the glycerinated virus. For these reasons dry points are no longer permitted in interstate traffic in accordance with the federal regulations.

The superiority of the glycerinated virus will be evident from a study of the ripening or purification of vaccine virus with glycerin (see below).

The old-fashioned dry points must not be confused with the points now placed on the market by manufacturers containing a drop of glycerinated lymph. There is no special objection to these, except that it encourages vaccination by the method of scarification. Some manufac-

turers imitate the old-fashioned dry point by removing most of the glycerin from the ripened pulp by pressing it between blotting papers. The remaining pulp is then attached to the points with sterile dextrose, blood serum, or some other gummy substance.

The Process of Ripening.—When the vaccine virus is fresh it is said to be "green." Glycerin is added to the green pulp, and after it has acted a certain period of time the virus is said to be "ripe." The use of glycerin for this purpose was introduced by Moncton Copeman in 1891 for the purpose of preserving and purifying the virus. The glycerin acts as a differential germicide, that is, it preserves[1] the active principle in the vaccine virus, but destroys the frail non-spore-bearing bacteria. In time the virus itself succumbs. Vaccine virus must, therefore, not be used while green nor when too old. Manufacturers usually date their products as "not reliable after" or "return after" 4 to 6 weeks in the summer time and 3 months during the cold season.

Sixty per cent. glycerin of the best quality is used. I have shown that no growth of bacteria, yeasts, or molds takes place in this percentage. Two to four parts of 60 per cent. glycerin are added to 1 part of the pulp by weight. The mixture is then thoroughly ground with a mortar and pestle by hand, or between glass rollers in a special mill driven by machinery. The pulp should be thoroughly broken up and a uniform suspension obtained. The amount of glycerin added depends upon the consistency and character of the pulp. The only objection to adding more glycerin would be the greater dilution of the virus, and, therefore, a larger proportion of negative takes. A higher percentage than 60 per cent. of glycerin soon renders the virus inert. The glycerin probably destroys the bacteria by virtue of its dehydrating action. The time required for the virus to ripen depends upon the temperature. Most of the non-spore-bearing bacteria perish in 30 days at 15° to 20° C. Approximately the same effect may be obtained at 37° C. in from 24 to 48 hours. At low temperatures the glycerin has practically no bactericidal effect. The process must always be controlled bacteriologically.

Substances other than glycerin are used for the purpose of purifying vaccine virus. Carbolic acid (0.5 to 1.0 per cent.) is used with success in Japan, and to some extent in this country. Potassium cyanid, chloroform, chlorobutanol, etc., have been tried, with less success in practice.

Bacteria in Vaccine Virus.—Vaccine virus always contains bacteria. There is no such thing as aseptic vaccine virus. The active principle has not been grown in pure cultures. However, the bacteria which contaminate vaccine virus are, for the most part, harmless to man. They are commonly those that are found on and in the skin of the calf. The

[1] Glycerin also serves as a preservative for other filterable viruses, as foot and mouth disease, anterior poliomyelitis, rabies, etc.

non-spore-bearing varieties are largely eliminated by the process of ripening. There are fewer bacteria in the typical unbroken vesicle than in a broken, crusty, inflamed eruption. Green virus may contain from a few thousand to over a million bacteria per cubic centimeter. The ripened, glycerinated virus contains comparatively much fewer, and these mostly spores of the hay bacillus, common molds, and other harmless saprophytes. The number of such bacteria in the ripened virus may be taken as an indication of the care and cleanliness with which the virus has been prepared. Staphylococci, streptococci, members of the hemorrhagic septicemic group, and, in a few instances, tetanus spores and the gas bacillus have been found in vaccine virus.

Seed Vaccine.—The seed virus may be obtained (1) from cowpox, (2) from smallpox, (3) by retrovaccination.

Spontaneous or casual cowpox occasionally occurs, that is to say, the disease appears to arise spontaneously because its origin cannot be traced. Casual cowpox comes either from another case of cowpox or from a case of smallpox. Cattle are not subject to smallpox, but, when smallpox virus is introduced into the skin of a calf, it produces cowpox. When smallpox is thus converted into cowpox, it remains fixed as such, and never reverts to smallpox.[1] In several instances in England, Germany, and this country the seed virus has been obtained by starting cowpox through the inoculation of smallpox virus. Such virus should not be used until several transfers from calf to calf have been made, for the reason that some of the smallpox virus may be carried over unaltered, during the first few transfers. Retrovaccination consists in carrying the vaccine virus back from child to calf. By this method its virulence is maintained. Instead of calves, monkeys or rabbits may be used for the purposes of retrovaccination.

Propagation.—In the propagation of bovine virus young calves are preferred, because they are more manageable, the skin is more tender, and the eruption is therefore more abundant and typical. With young animals a milk diet may be used, which simplifies the problem of dust contamination from dry feed. If hay or fodder is used, it must first be autoclaved. Either heifers or bull calves are suitable.

The animals are held in quarantine for seven days, under observation, to determine the absence of infections such as tuberculosis, glanders, foot-and-mouth disease, tetanus, and skin eruptions of any kind.

Before vaccinating the calf it is carefully cleaned, and the site of the inoculation is shaved and prepared surgically, but without the use of germicidal solutions. Germicides are not suitable for the reason that they are apt to destroy the vaccine virus. Cleanliness and asepsis are the watchwords. The area selected is usually the abdominal wall be-

[1] It is highly significant that casual cowpox was formerly much more common when smallpox was much more prevalent.

tween the tip of the sternum and the groin, sometimes including the inner side of the thigh. The usual method is to make long, superficial incisions in the skin about one inch apart, and the seed virus is gently rubbed into these incisions. The calves must then be kept rigidly isolated in a special room, moderately lighted, free from dust, and screened to keep out insects. The temperature of the animal is taken several times daily, and the eruption at each stage of the disease is closely watched and recorded.

The virus is usually taken from the animal between the fifth and the eighth day. It is an advantage to take the virus as early as practicable, in order to avoid contaminating infections which may occur when the vesicles suppurate. Only typical and entirely characteristic vesicles should be removed. Before the virus is removed, the animal is killed to avoid pain, and an autopsy is done as soon as the virus is removed. If the autopsy shows any lesions indicating infections other than vaccinia, the virus is discarded.

It is not wise in propagating vaccine virus to vaccinate too large an area. This favors infections by lowering resistance; less typical eruptions are obtained than when the area vaccinated is moderate in extent. A yield of from twenty to forty grams of pulp from one calf should satisfy the propagator.

Before the virus is taken the animal is placed upon a special table, the site of the vaccination exposed and given a very thorough washing and prolonged scrubbing with soap, and an abundant flushing with sterile water. The pulp is usually obtained by scraping the vesicles with a sharp spoon curette.

Glycerin (60 per cent.) in proper proportion is added at once to the pulp, and this is ground to a state of fine and uniform subdivision in a Döring lymph mill, or simply by hand with a mortar and pestle. This glycerinated pulp is then allowed to ripen, and when ripe it is hermetically sealed in capillary tubes, or placed in small vials, or upon glass or ivory points, for the market.

METHODS OF VACCINATION

Vaccination consists in transferring the virus from one animal to the skin of another animal. The operation may be compared to the transfer of a culture in a bacteriologic laboratory. Precisely similar precautions to prevent contamination must be used in both cases. Vaccination must be regarded as a surgical operation. No person unfamiliar with surgical cleanliness should be permitted to perform this "little" operation.

The vaccine virus may be introduced in one of three ways: (1) by puncture, (2) by incision, or (3) by scarification.

Jenner used punctures or short incisions. Later blisters were raised upon the skin and the virus placed upon the abraded surface. The incisions were then increased in number, and finally cross scratchings were made.

Puncture.—The simplest and best method is puncture with a needle, for there is least chance of contamination and the eruption is typical. The disadvantage is that the virus now used is diluted with glycerin, and therefore somewhat attenuated, so that a few simple punctures are less apt to take.

Incision.—The method advised and recommended is that of incision. Incision is the only method of vaccination permitted by the laws of Germany, and recommended by the Local Government Board of England. Incision, if not too deep, consists really of a series of punctures, and serves the same purpose. Incisions may be made with the point of a scalpel. I prefer to use a needle. The incision or scratch should not be deep enough to draw blood, but a few drops do no harm. It is rather difficult to control the depth of the incision with a scalpel, especially if it is sharp. Scratching with a needle is much more easily controlled. The incisions should be about three-quarters of an inch long and about an inch apart. The vaccine virus is then placed upon the abraded surface, and gently rubbed, not ground, in. It is important not to cause any unnecessary irritation so as to avoid attracting infections.

Scarification.—Scarification or cross-scratching is prohibited in Germany by ministerial decree of March 31, 1897, which was incorporated into the revised rules of the Bundesrath, July 28, 1898. The objection to scarification is that this method produces an abraded surface which is soon covered by a crust of serum and blood, through which the eruption cannot pierce. The vesicles form a ring around the scarified area, leaving a central irritated wound, inviting infection. It is believed that most of the cases of tetanus complicating vaccination occurred in cases in which scarification was used. In this method favorable anaerobic conditions are produced under the crust or scab which forms over the abraded surface.

The Point of Election.—The outer surface of the left arm at about the insertion of the deltoid is the most convenient for the operator and the patient. This is the original site selected by Jenner, and is less liable to severe glandular complications than other points.

Flachs recommends the side of the chest at about the level of the sixth rib, in the axilla. Here the scar is not visible; there is little motion, and it is easily bandaged, but this site is open to the disadvantage of greater heat and moisture and there is, therefore, greater danger of complications.

The leg is sometimes selected to avoid disfigurement. The vaccina-

tion scar should not be regarded as a deformity. To the sanitarian a typical vaccine scar is a sanitary dimple. The leg is more exposed than the arm to traumatism, and, therefore, to complications. Dock refuses to vaccinate on the leg unless the patient will stay in bed until the vesicle heals. With babies in diapers and with young children it is exceedingly difficult to keep these parts clean. If the leg is selected, the vaccination should be done on the calf below the head of the fibula, and not on the outer surface of the thigh.

Number of Incisions.—This has an important bearing upon the probability of the take, as well as the protection. It is not wise to depend upon one. The relation of the number of vesicles and the amount of reaction to the degree and length of the immunity has not been worked out. The German regulations of 1899 require at least four incisions, each one centimeter long, and two centimeters apart. The Local Government Board of England directs that four vesicles should be produced, and that the total area of the vesicle formation shall not be less than one-half a square inch. My own practice follows that of Dock, who makes not less than two incisions about an inch long and an inch apart; but in case of exposure to smallpox three or four such incisions are advisable.

The Operation.—The skin at the site of the operation must be surgically clean, but need not necessarily be treated with antiseptics. If such are used, they must be carefully washed away in order not to destroy the activity of the virus. A thorough scrubbing with soap and water is necessary for a dirty skin. Washing with warm water followed by alcohol is usually enough. The alcohol should be permitted to evaporate before the incision is made. In general, the less the skin is irritated the less is the danger of complications. Needles are particularly handy, as they may be flamed just before the operation, and are convenient in saving time when many people are to be vaccinated. The vaccine virus is gently rubbed into the incision, not ground in, and then allowed to dry. No dressing is necessary at the beginning, but several layers of dry sterile gauze held in place by adhesive plaster do no harm, and serve as a protection. Pads, plasters, and shields of any sort are unwise, because by retaining heat and moisture they cause a softening and breaking down; in other words, they act like a poultice. Bathing need not be omitted, nor any of the ordinary occupations, but unnecessary use of the arm must be guarded against, as this increases the congestion, inflammation, and the chances of infection.

Schamberg and Kolmer[1] have recently advised the use of a 4 per cent. alcoholic solution of picric acid on the vaccinated area 48 hours after the insertion of the lymph. This apparently does not interfere with the success of the vaccination. Schamberg and Kolmer believe

[1] *Lancet*, Nov. 8, 1911, CLXXXI, No. 4603.

that the picric acid lessens the degree of the local inflammatory reaction and that the patients are not so apt to exhibit constitutional disturbances. It also decreases the liability of extraneous bacterial infection.

INDICES OF A SUCCESSFUL VACCINATION

The take must be typical and the clinical course characteristic, otherwise we have no assurance that the individual is protected against smallpox. The best indices of a successful take are: (1) the course of the eruption, (2) the general symptoms, and (3) the scar.

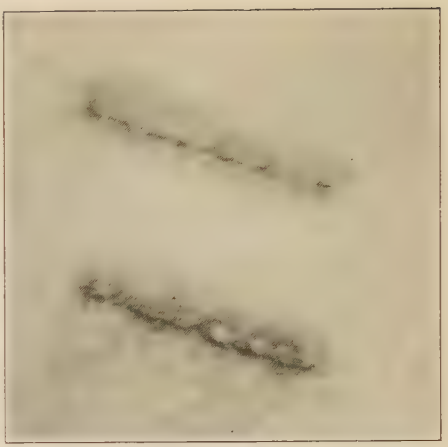
The importance of knowing the skin lesions of vaccinia were insisted upon by Jenner. Every vesicle, scab, ulcer, or irritated wound is not vaccinia. No confidence should be placed in doubtful or atypical takes. The typical features of vaccination are singularly alike. The clinical course of a primary vaccination is as follows:

Course of the Eruption.—The primary wound soon heals. Apparently nothing occurs for 3 to 4 days, which is the period of incubation. Then one or more small papules appear upon the skin where the vaccine virus was introduced. The papule is small, round, flat, bright red, hard, but superficial. About the fifth day the summit of the papule becomes vesicular. The vesicle is at first clear and pearl-like. Umbilication soon develops as the vesicle enlarges. A deep, red, and swollen areola surrounds the vesicle and grows wider as the lesion advances. This gives the picture of the "pearl upon the rose leaf" which constitutes the true Jennerian vesicle. By the seventh day the vesicle is full size, round or oval, flat on top, umbilicated, and contents clear. It is multilocular; if pricked with a pin or accidentally opened only that portion of the lymph contained in the compartment opened will exude. By the eighth day it turns yellowish, the middle is fuller, following which the so-called second umbilication develops. Meanwhile the areola deepens, widens, and may be swollen. The skin feels hot, is painful, and the axillary glands become enlarged and tender. About the ninth day the areola begins to fade and the swelling subsides. By the eleventh or twelfth day the vesicle rapidly dries, leaving a brown, wrinkled scab, which finally drops off. It should never be removed, as it forms the best bandage.

The scar is at first red, finally turns white, with the pits or foveations so characteristic of true cowpox.

General Symptoms.—The general symptoms vary. There are malaise, loss of appetite, sometimes nausea and vomiting, headache, pain in the muscles of the back, and other indications of a mild febrile reaction. The temperature may go to 38° or 38.5° C. between the third and seventh days. The febrile reaction bears no special relation to the size and number of the vesicles or to the areola. The nitrogen elimination

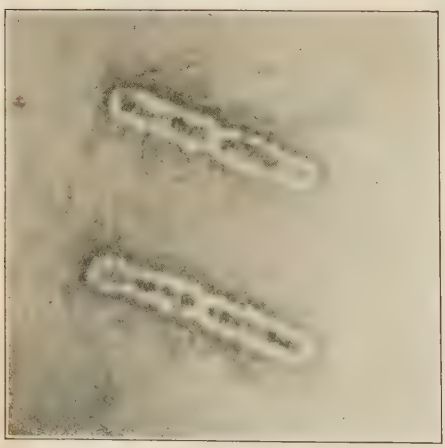
Fourth Day



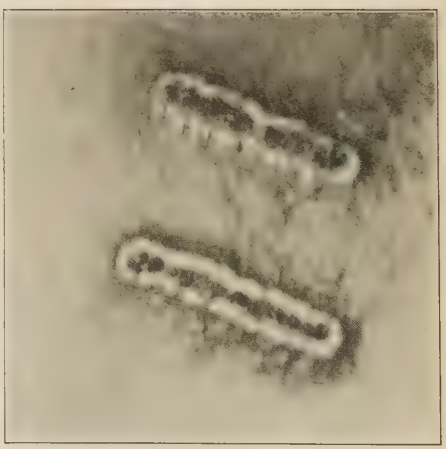
Fifth Day

Sixth Day

Seventh Day



Eighth Day



Ninth Day

FIG. 1.—VACCINIA. COURSE OF THE ERUPTION FROM THE FOURTH TO THE NINTH DAY.

Tenth Day

Eleventh Day

Twelfth Day

Thirteenth Day

Fourteenth Day

Scar—Sixth Week

Fig. 2.—Vaccinia. Course of the Eruption from the Tenth Day.

increases about the tenth day for a short time. The blood changes resemble those of smallpox, an early leukopenia and secondary leukocytosis.

Secondary vaccinations often run an accelerated, milder, or modified course with shortened periods of incubation (see revaccination).

THE IMMUNITY

The immunity appears about the eighth day of the vaccination. Layet puts the point of safety at the ninth day, Burckhard at the eleventh. These data are based upon the early work with variolation, when persons were inoculated with smallpox at various periods following vaccination. Sacco got only local eruption by inoculating smallpox on the eighth to the eleventh days, and none after that.

Vaccination protects not only against smallpox, but also against vaccinia. Curiously enough, the degree and length of immunity appear to be greater against smallpox than against itself. It is irrational to attempt to fix a definite time for the duration of the immunity. This varies as in other infectious processes. It is known through experiment and experience that the immunity gradually wears off. Definite protection on the average lasts about seven years. The degree of protection is usually absolute for some years, and then gradually fades. In this, as in other diseases, immunity is a relative term. Smallpox itself does not always protect against smallpox. Some people have two and even three attacks of smallpox. Such cases, however, are exceptional, and it is also exceptional to have smallpox occur in an individual who has been properly vaccinated.

Careful statistics collected in Japan since 1879 show quite definitely the gradual diminution of the immunity, beginning with the second year after vaccination. Kitasato's table,[1] based on 951 cases, is as follows:

SUCCESSFUL REVACCINATION AFTER:	
1 year	13.6 per cent.
2 years	32.9 " "
3 years	46.6 " "
4 years	57.3 " "
5 years	51.1 " "
6 years	63.8 " "

Weil, in 1899, reported 72.5 per cent. of successful revaccinations after seven years, 80 per cent. after eight years, 85 per cent. after nine years, and 88.6 per cent. after ten years.

It is a fallacy to state that, if a revaccination takes, the subject was therefore susceptible. While this is usually true, it does not necessarily

[1] *Journal A. M. A.*, March 25, 1911, p. 889.

follow. It is a still greater fallacy to state that, if a vaccination fails, the subject is therefore immune. This view may result in real harm. Vaccination may fail for many reasons—the operation may not have been properly done, or the virus may have been inert. Sometimes persons are unsuccessfully vaccinated three, four, or more times before a typical take is obtained.

The nature of the changes in the body which produce the immunity are not understood. In this sense vaccination is still an empiric procedure. We now know of many analogous instances, however, where an active acquired immunity is induced by means of an attenuated virus. The immunity produced by vaccine virus does not depend upon an antitoxin. The blood, however, contains specific antibodies, shown by the fact that equal parts of blood serum from a calf two weeks after successful vaccination mixed with vaccine virus destroy its activity.

REVACCINATION

The fact that the immunity wears off after a number of years makes it necessary to practice revaccination in order to afford a continuous protection. There is some difference of opinion as to just when it is best to vaccinate the second time. Ten years is too long a period, probably, to depend upon in individual cases. One year—advised by some—is shorter than necessary in most cases. The five-year interval of Japan is good in many respects, but probably not better than revaccination in the twelfth year obligatory in Germany.

The best time to vaccinate is in the first year before the second summer, again at from ten to thirteen years. After this it is usually unnecessary to vaccinate again, unless there is particular danger of exposure to smallpox.

All persons exposed directly or indirectly to smallpox should at once be vaccinated—unless they have had the disease or have recently been successfully vaccinated. There are no contraindications to vaccinating babies immediately after birth.

The clinical picture of secondary vaccinations may be quite different from the typical take following a primary vaccination. These altered reactions were known in the time of Jenner, but were lost sight of until recently rediscovered, and their significance realized from studies in anaphylaxis.

Revaccinations may be divided into three groups: (1) they may run an unaltered course resembling primary takes in all respects, showing that immunity to cowpox has disappeared; (2) they may run a slightly more rapid course in which the period of incubation is shortened and in which the height of the pustular stage occurs about the sixth day (this is known as the accelerated reaction); or (3) they may run a

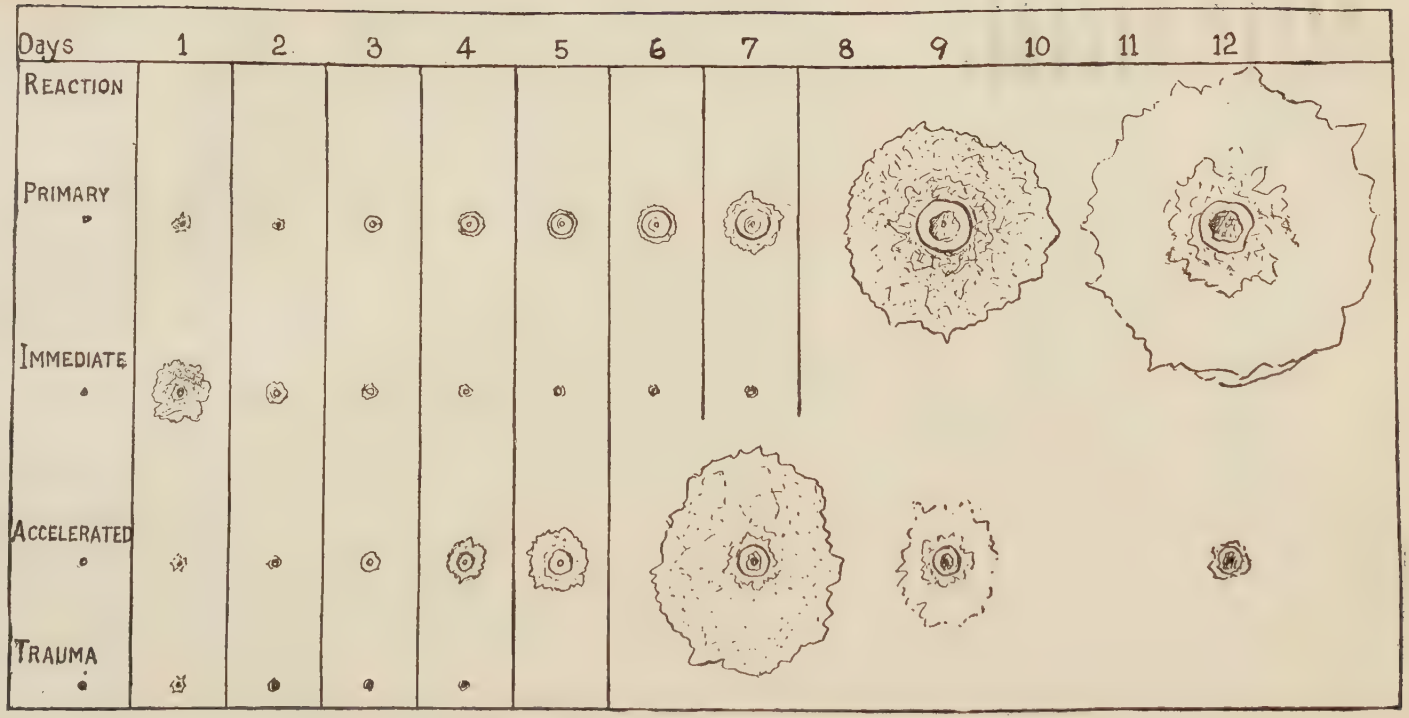


Fig. 3.—A. Course of Primary Vaccination with Cowpox, from Day to Day.
B. Revaccination after a Short Interval—Early or Immediate Reaction.
C. Revaccination after a Long Interval—Accelerated Reaction.
D. Traumatism Alone. [Modified from von Pirquet, *Arch. Int. Med.*, Feb. and Mar., 1911.]

very much shortened, milder, and rapid course. The eruption may be only a small papule or an almost imperceptible erythema which soon disappears; the period of incubation is less than 24 hours. This is known as the immediate reaction and resembles a cutaneous tuberculin reaction in many respects. These altered reactions have been studied especially by Von Pirquet and are shown graphically in Fig. 3.

The immediate reaction may be put to practical use in order to distinguish smallpox from chickenpox. Thus, Tièche has shown that smallpox virus introduced into the skin of a person immunized by vaccination will show the typical immediate reaction; whereas the virus of chickenpox is invariably negative. This test can be freed of all possible danger by heating the virus to 60° C. for 30 minutes, which does not seem to affect the reaction.

CLAIMS FOR VACCINATION

1. If successful, it protects the individual against smallpox for a period which has not been determined mathematically for the individual, but which averages about seven years.

2. The protection may be renewed by a second vaccination.

3. Persons successfully vaccinated on two occasions are usually immune against smallpox for life.

4. Vaccination and revaccination systematically and generally carried out confer complete protection to a community or a nation. In other words, while the individual protection is not always perfect, the communal protection is absolute.

5. A person vaccinated once and at a later time contracting smallpox as a rule has the disease in a less serious form than unvaccinated persons (varioloid).[1] The degree of favorable modification of smallpox is in inverse proportion to the period of time elapsing between the vaccination and the attack of smallpox.

6. The beneficial effects of vaccination are most pronounced in those in whom the vaccine affection has run its most typical and perfect course.

VACCINATION OF EXPOSED PERSONS

The question frequently arises whether persons exposed to smallpox should be vaccinated. The effect of vaccination during the period of incubation of smallpox is very interesting, and may be summed up as follows:

[1] The term varioloid was introduced by Thompson in 1820 to describe the mild and modified form of smallpox occurring after vaccination. The eruption in varioloid disappears more rapidly than in variola. Yolfert, Dornbleuth, and Harden showed that one vaccination was not sufficient protection against smallpox for a lifetime, that revaccination was necessary and that the clinical manifestations of this vaccination are as different from those of the first vaccination as varioloid is from variola.

1. Vaccination just before or during the primary fever of smallpox does not influence the disease, nor does the vaccination take.

2. If the vaccination is done during the last stage of the period of incubation of smallpox, the two infections run their course side by side without influencing each other.

3. If it is done about the sixth or eighth day of the period of incubation the vaccination takes and may modify the severity of the smallpox.

4. Vaccination done at the beginning of the incubation period in time to have the vaccine eruption reach maturity before the smallpox begins will prevent or abort the disease. This is shown in the following diagram:

THE EFFECT OF VACCINATION DURING THE PERIOD OF INCUBATION OF SMALLPOX

On the First Day	Early in the Incubation Period 2nd to 6th days	Middle of the Incubation Period 6th to 8th days	Toward the end of the Incubation Period 9th to 14th days	During the Primary Fever, or Preëruption Stage	
Prevent smallpox	Smallpox is aborted	Varioloid or mild case	Smallpox not influenced	Smallpox not influenced	Variola
1	2 3 4 5 6	7 8 9 10	11 12 13 14	1 2 3	
	Period of Incubation of Smallpox—in Days			Primary Fever	Eruption
The vaccination takes.	The vaccination takes.	The vaccination takes 2 or 4 days before primary fever.	The vaccination takes and both affections run side by side.	The vaccination does not take (?)	Vaccinia
		To produce the best results the vaccination should precede this period, so as to reach maturity before the onset of the primary fever. The vaccine vesicle reaches maturity about the 8th day.			

As we can never be quite sure just what stage in the period of incubation a given case may be in, it is always advisable to vaccinate exposed persons. Furthermore, little harm will be done if it is too late and the vaccine eruption is added to the smallpox. Indeed, Hanna,[1] presents claims to the effect that there is abundant evidence in mitigating the severity of smallpox when vaccination is performed at any time after infection up to the day of onset and even afterward.

[1] *Public Health*, July, 1910, XXIII, No. 10, p. 351.

DANGERS AND COMPLICATIONS

The alleged danger from vaccination has been greatly magnified by the antivaccinationists. However, vaccination is not always a harmless procedure; it must be looked upon as the production of an acute infectious disease, and, although the disease is always mild and trivial, it must not be treated as trifling. The chief danger lies in the fact that we have produced an open wound, which is subject to the complications of any wound. Even a pin prick or a razor scratch may result in death. While the aggregate number of deaths resulting from the complications of vaccination may be considerable, the aggregate of the individual risk is so small as to be disregarded, especially when proper precautions are taken. Many of the infections after vaccination occur in those in whom the regard for cleanliness is slight, and who neglect the site of the wound. In recent years, owing to the improved quality of the vaccine virus and the introduction of aseptic methods, a bad sore arm is a rare occurrence, and serious complications still rarer. In any case, the danger connected with vaccination is infinitesimal when compared with the benefit conferred. The important complications are:

Auto Vaccination.—This is usually due to scratching the virus into the finger, the nose, the mouth, the mucous membranes, or any part of the skin. When carried into the eye it may cause blindness. Physicians sometimes vaccinate their lips by blowing into vaccine tubes. In vaccine establishments accidental vaccination of the hand is common.

Generalized Vaccination.—This is sometimes reported, but is usually a mistaken diagnosis. A generalized eruption of cowpox is exceedingly rare, if it ever occurs. I have seen it in the calf after intravenous injection of a large amount of the virus, in which case there is a prolonged period of incubation.

Wound infections, such as ulcers, gangrene, erysipelas, abscesses, lymphangitis, suppuration of the axillary glands, and other septic infections are now exceedingly rare, and should be treated with the usual measures to prevent their occurrence.

Impetigo contagiosa occasionally occurs and may be a serious complication of vaccination, especially the bullous impetigo or pemphigoid forms, which presumably have their origin in cattle.

Syphilis, tuberculosis, and leprosy are sometimes feared, but these are practically impossible with the use of bovine virus. In any case it is doubtful whether tuberculosis or leprosy could be so transferred.

Tetanus deserves a special word. Several outbreaks have been reported in this country after the use of certain viruses. Willson in 1902 found tetanus spores in the vaccine virus used in a New Jersey outbreak. Glycerin does not destroy the tetanus spore. Many hundreds of

examinations made in the Hygienic Laboratory at Washington have failed to discover a tetanus spore in a single vaccine point or tube. The occasional danger cannot be denied. It is probable, however, that the infection in some of these cases comes from outside sources.

The occurrence of occasional stray spores in vaccine virus was demonstrated by Carini.[1] Such vaccine, however, had proved entirely harmless in thousands of cases. It is more than probable that the actual danger would begin if such occasional stray spores were allowed to germinate in the vaccine pulp through some serious fault in manipulation. It is conceivable that the vaccine pulp after removal from the calf or heifer, if not at once chilled, or if not at once mixed with glycerin, may form a very rich medium for anaerobic bacteria. Some carelessness or neglect just at this stage might prove disastrous if tetanus spores accidentally present should multiply. The epidemic in this country in 1902 reported by Willson[2] and MacFarland[3] may have been the result of some such occurrence. On the other hand, neglected vaccination wounds or those in which proper bandages or shields favor anaerobiosis may stimulate the germination of spores coming from without and lead to the occasional reported sporadic cases following vaccination.

To prevent tetanus complications it is important to avoid scarification and irritation, also to avoid the use of shields and bandages which favor anaerobic conditions; to practice strict cleanliness, and to use vaccine virus that has been properly prepared and tested. Special tests for tetanus are now required by federal regulations of every lot of vaccine virus before it is placed upon the market.

Foot and Mouth Disease.—The infection of foot and mouth disease has in one instance been demonstrated as a contamination of vaccine virus.[4] It is, however, impossible to convey foot and mouth disease to man through cutaneous inoculation. While no harm has been done to man, the contamination is undesirable, and special federal regulations now require vaccine virus to be tested from time to time to assure its freedom from this infection.

As an illustration of how seldom complications are caused by vaccination we have the results of Germany, where in thirteen years (1885-1893) 32,166,619 children were vaccinated. Of these 115 died within a few weeks or months after the operation, presumably of injuries incidental thereto. Of these at least 48 probably did not die as a direct result of the vaccination.

The figures of recent years are still better, for it is now exceedingly rare for a death to be recorded as directly due to vaccination. For

[1] *Centralbl. f. Bakt.*, Orig. 1904, XXXVII, p. 1147.
[2] *Jour. A. M. A.*, 1902, XXXVIII, p. 1147.
[3] *Jour. Med. Research*, 1902, n. s. II, p. 474.
[4] Mohler and Rosenau, U. S. Dept. of Agriculture, B. A. I. Circular 147, June 16, 1909.

example, in the Philippine Islands in the past few years the United States authorities vaccinated 3,515,000 persons without a single death or any serious post-vaccinal complications.

THE GOVERNMENT CONTROL OF VACCINE VIRUS

By the law of July 1, 1902, the vaccine virus sold in interstate traffic in the United States must come from a licensed manufacturer. These licenses are issued by the Secretary of the Treasury only after a careful inspection of the plant, personnel, and product by a competent government officer. The licenses are good for one year only, and are reissued only after reinspection. The government regulations require each lot of vaccine virus to be examined carefully by modern bacteriological methods to determine the number of bacteria, and special tests must be made to determine the absence of pathogenic microorganisms. These tests include animal inoculations, as well as cultural methods. Special tests for each lot of vaccine must be made to determine the presence or absence of streptococci, tetanus spores, foot and mouth infections, etc. The government does not guarantee the purity and potency of each package of vaccine virus, but through its inspections and frequent examinations of the virus on the market every confidence may now be had in the vaccine virus propagated by licensed manufacturers in this country.

THE UNITY OF COWPOX AND SMALLPOX

The unity or duality of these two diseases has been the subject of much contention. Jenner originally considered cowpox to be a modified smallpox. The successful experiments in Germany, England, and this country, in which smallpox has actually been modified by passing variolous matter through calves has proved positively that we are dealing with two forms of one disease. Much of the vaccine virus used during the past hundred years was originally obtained from cases of casual cowpox. This virus has been shown by experience and experiments to protect against smallpox, which makes it highly probable that we are dealing with one disease. The parasite *Cytorrhyctes variolæ*, discovered by Councilman, Brinckerhoff, and Tyzzer, gives a probable explanation of how smallpox may, under certain circumstances, become attenuated. The life cycle of this parasite interpreted by Calkins indicates that the mild disease, cowpox or vaccinia, is due to the asexual phase in the life cycle of the parasite which lives and multiplies in the cytoplasm of the epithelial cell; smallpox is caused by the combined asexual and sexual cycle of the same parasite, the latter phase occurring in the nucleus of the epithelial cell. When the *Cytorrhyctes variolæ* loses its power to generate by sexual division it never again regains it; that is, while small-

pox may be modified into cowpox, cowpox has never been returned to smallpox.

It seems plain that much of the so-called casual cowpox probably has its origin from smallpox through accidental inoculation in milking or handling these animals by persons having or recovering from variola. Once started, the propagation of the modified virus from cow to cow would be comparatively simple.

COMPULSORY VACCINATION

Vaccination affords a high degree of immunity to the individual, and a well-nigh perfect protection to the community. To remain unvaccinated is selfish in that such persons steal a certain measure of protection from the community on account of the barrier of vaccinated persons around them.

The laws[1] and regulations relating to vaccination in the several states of the United States show marked lack of uniformity. Compulsory general vaccination can be said to exist by law only in Kentucky, Rhode Island, and Porto Rico.[2] Arizona, Hawaii, Maryland, New Mexico, North Dakota have laws requiring vaccination of children. In recent years smallpox has been so mild in the United States that the case death rate has been as low as 0.2 per cent., or 1 death in 500 cases.

Decisions in the various courts in the United States have held compulsory vaccination to be legal. A decision of the Supreme Court of the United States (Henning Jacobson vs. The Commonwealth of Massachusetts, April 1, 1905) upheld in every respect the statute, the validity of which was questioned under the Constitution.

> The liberty secured by the Constitution of the United States . . . . does not impart an absolute right in each person to be, at all times, and in all circumstances, wholly freed from restraint. Real liberty for all could not exist under the operation of a principle which recognizes the right of each individual person to use his own, whether in respect to his person or his property, regardless of the injury that may be done to others.

Theoretically it would be ideal if all persons submitted to vaccination and revaccination voluntarily. But experience has shown that this is impractical, and, wherever tried, has failed. The best results have always been obtained where vaccination has been compulsory, and, in my judgment, this is the only present means by which smallpox may be eliminated.

The world may learn a valuable lesson from the splendid results obtained in Germany through compulsory vaccination and revaccina-

[1] Kerr, J. W., "Vaccination, and Analysis of the Laws and Regulations Relating Thereto in Force in the United States," *Public Health Bull. 52.*

[2] Massachusetts, in 1809, was the first state to enact legislation relative to vaccination.

tion. In England the "conscience clause" allows many persons to remain unvaccinated and thereby seriously diminishes the effects of the vaccination laws of that land. In Minnesota the state health authorities became weary of the clamor against compulsory vaccination and assisted in having the law repealed. They said, in substance, to the people of the state: "Take your choice. Be vaccinated and protect yourself, or run the risk of contracting smallpox; if you get it, it is your own fault."

TABLE 1.—DEATHS FROM SMALLPOX IN COUNTRIES WITH COMPULSORY VACCINATION AND THOSE WITHOUT COMPULSORY VACCINATION

Population	Smallpox Deaths				Average of Deaths	Average per Million of Population
	1886	1887	1888	1889		
Sweden*..... 4,746,465	1	5	9	2	4	1
Ireland*..... 4,808,728	2	14	3	0	5	1
Scotland*..... 4,013,029	24	17	0	6	12	3
Germany*..... 47,923,735	197	168	112	200	169	3.5
England*..... 28,247,151	275	505	1,026	23	458	16
Switzerland..... 2,922,430	182	14	17	3	54	18.5
Belgium..... 5,940,365	1,213	610	865	1,212	975	164
Russia..... 92,822,470	16,938	25,884	?	?	21,411	231
Austria..... 23,000,000	8,794	9,591	14,138	12,358	11,220	510
Italy..... 29,717,982	?	16,249	18,110	13,416	15,925	536
Spain..... 11,864,000	?	?	14,378	8,472	11,425	963

*Compulsory vaccination.

INOCULATION OR VARIOLA INOCULATA

The practice of inoculation must be carefully distinguished from that of vaccination. By inoculation we mean the introduction of smallpox matter into the skin of man. The disease thus produced is usually very mild, but is nevertheless true smallpox, and just as contagious as smallpox.

This phase of the subject may be made clearer by considering smallpox as existing in three forms: (1) *variola vera* or true smallpox; (2) *variola inoculata* or inoculated smallpox; (3) *vaccinia,* cowpox, or modified smallpox. The differences between these affections are shown in the table on the following page.

Emphasis must be placed on the fact that variola inoculata, while usually a mild disease, is just as communicable as true smallpox, and those who contract the disease in this way get true smallpox, often in serious or fatal form. *Inoculation, therefore, protects the individual but endangers the community.*

Variola Vera	Variola Inoculata	Vaccinia or Cowpox
True smallpox.	Inoculated smallpox.	Modified and attenuated smallpox.
Only occurs in man.	Occurs in man and monkeys.	Man, monkeys, cattle, guinea-pigs, rabbits, rats, camels, and many other mammals.
High mortality.	Milder; rarely fatal; about 1 in 500.	Very mild; never fatal.
A general eruption, often confluent or hemorrhagic.	A general eruption, fewer pustules (rarely over 200); seldom confluent or hemorrhagic.	Always local and confined to the site of the vaccination.
Highly contagious	Equally highly contagious.	Not contagious — contracted only by mechanical transfer of vaccine virus.
Period of incubation 12-14 days.	8 days.	3-4 days.

Inoculation is a very old custom. It was practiced by the Chinese from time immemorial. The method was introduced into western civilization through Lady Mary Wortly Montagu, who learned of the method at Constantinople and had her own boy "engrafted" with successful result. In 1717 Lady Montagu wrote her now famous letter to her friend Sarah Chiswell, and the practice soon became popular in England (1721) and spread to America and the Continent. It was introduced into this country by Dr. Zabdiel Boylston at Boston. But the dangers were early realized and inoculation was soon replaced by vaccination. According to Plehn, inoculation is still practiced in central Africa.

The method of inoculation is precisely similar to that of vaccination. The matter is obtained from the vesicle or pustule of a case of smallpox. This material is then introduced into the skin by means of a puncture, an incision, or through an abraded surface. The Chinese, it is said, practice inoculation by blowing the dried smallpox crusts into the nostrils.

While inoculation has properly fallen into disuse, there are conceivable emergencies in which the practice would be justified. For example, on board ship or on an island or isolated place, in the absence of vaccine virus. Under such circumstances it would be essential to inoculate everybody at the same time.

The inoculation of smallpox will always remain for the student of preventive medicine one of the most interesting episodes in the develop-

ment of the sanitary sciences. It illustrates in the clearest manner some of the fundamental phenomena of infection, susceptibility, and immunity. It was animal experimentation on a huge scale, the like of which we shall never see repeated on man as the subject (Sedgwick). It is now a matter of regret that for the sake of science better advantage was not taken of the data.

PREVALENCE OF SMALLPOX

It is very difficult for us now to realize that smallpox was once much more common than measles and much more fatal. Many of those who recovered were disfigured for life, left blind, or with some other serious consequence of the disease. For centuries smallpox was one of the greatest scourges. It depopulated cities and exterminated nations. In Europe alone, where its ravages were comparatively slight, it killed hundreds of thousands yearly. In the 18th century, of which we have the best records, almost everybody had it before he grew up. Parents sometimes exposed their children to the disease in order to be through with it, just as they now sometimes do with the minor contagious diseases.

Smallpox was formerly a disease of children. It was called *"kinderblättern."* Since vaccination protects the child, smallpox has now become more prevalent among adults.

The distinguished mathematician, Bernouille, estimated that 15,000,000 people died of smallpox in 25 years in the 18th century. It has been estimated that 60 million people died of smallpox during that century. Haygarth gives an account of a smallpox epidemic in Chester, England, population 14,713. At the termination of the epidemic there were but 1,060 persons, or 7 per cent. of the population, who had never had smallpox. Many similar instances are cited in the literature. The French physician de la Condamine (1754) said that "every tenth death was due to smallpox and that one-fourth of mankind was either killed by it or crippled or disfigured for life." Sarcone (1782) estimated the number of persons in Italy who suffered from smallpox as 90 per cent. of the population.

Smallpox was introduced into the western hemisphere by the Spaniards about 15 years after the discovery of America. In Mexico within a short period three and one-half million persons are said to have died of the disease (Chapman). Catlin (1841) states that of 12,000,000 American Indians 6,000,000 fell victims to smallpox. In Iceland, in 1707, 18,000 perished out of a population of 50,000, that is, smallpox killed 36 per cent. of the total population in one year.

A good example is that of Boston in 1752, population at that time 15,684. Of this number 5,998 had previously had smallpox. During the epidemic 5,545 persons contracted the disease in the usual manner,

and 2,124 took it by inoculation. 1,843 persons escaped from the town to avoid the infection. There were, therefore, left in the city but 174 persons who had never had smallpox.

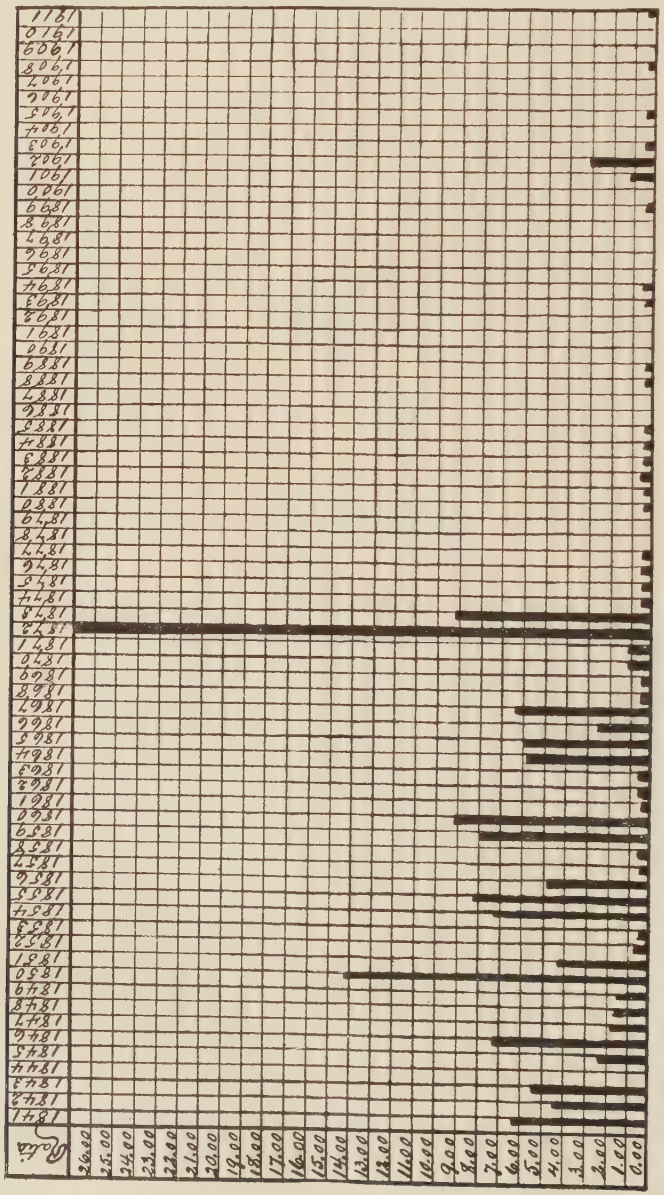


FIG. 4.—RATIO OF MORTALITY OF VARIOLA PER 10,000 OF THE POPULATION, IN BOSTON, FROM 1841 TO 1911, INCLUSIVE.

Average Population in Boston from 1841 to 1873, Inclusive, 172,141. Number of Deaths from Variola from 1841 to 1873, Inclusive, 2834 Average Population in Boston from 1874 to 1911, Inclusive, 484,273. Number of Deaths from Variola from 1874 to 1911, Inclusive, 343.

Smallpox is still as serious as it was in former times. Thus, in five years, from 1893-1897, 346,520 persons died of smallpox in sixteen

countries. Of this number Russia alone lost 275,502. These figures are the more terrible when it is realized that these lives might have been saved by the use of a simple prophylactic measure within reach of all.

EPIDEMIOLOGY

Few of the acute infectious diseases show such a complete independence of conditions such as race, climate, soil, age, sex, and occupation, sanitary surroundings, etc., as does smallpox. It thrives wherever the contagion is carried, and wherever it finds susceptible people. Probably no one is naturally immune. The susceptibility of the population varies, because a smallpox outbreak leaves so many immune. This is one reason why the disease recurs in waves. The mortality varies greatly in different epidemics. At times it is less than one per cent.; it frequently reaches thirty per cent. and over.

In 1901-1903 the mortality in the United States was as low as 2 per cent., and following that 0.5 per cent. These differences occurred in the prevaccination era as well as now.

The epidemiology of smallpox bears no relation to improved sanitation, which has diminished the prevalence of tuberculosis, typhoid, cholera, and has practically subdued typhus and relapsing fever. It is evident that general sanitation could not affect contagious diseases like smallpox and measles. Smallpox spares neither high nor low, the rich or poor; before the days of vaccination it counted many kings, queens, and princes among its victims.

MODES OF INFECTION

We are still ignorant of the precise mode by which smallpox is conveyed. The view generally held is that the infection is air-borne and enters the system through the respiratory mucous membrane. It has been surmised that a local lesion may be produced in this favorable soil, the so-called "propustule," from which general infection through the blood takes place. The blood infection is marked by a sharp onset (the initial symptoms), and the skin eruption is embolic in character. The objection to this view is that a careful search of 54 cases in Boston by Councilman and his colleagues failed to find such a propustule.

It is known that the Chinese inoculated the disease by placing a crust from the eruption in the nostrils, but whether the disease so produced was variola vera or variola inoculata is not known.

The virus of smallpox is always contained in the skin lesions. Of this we have experimental evidence. It is also supposed to be in the expired air. This, however, has never been experimentally proven. The disease is contagious before the eruption appears. It is even believed to be communicable during the period of incubation. Smallpox

has always been taken as the type of the contagious diseases; the contagion is the most "volatile" of any of the diseases of man. This volatility, however, has been overestimated, and, while probably an air-borne infection, the radius of danger is contracted. English observers have long taken the view that smallpox may be blown for great distances, and they attribute the prevalence of smallpox to the windward of hospitals as an indication that the virus may be carried down the wind. My experience with the disease teaches me that the danger from such a source is practically nil. One may safely live next door to a smallpox hospital that is well screened and properly managed. The influence of flies and other insects, or surreptitious visiting, may account for the spread of this disease outside of hospital walls.

In addition to more or less direct contact smallpox may be spread indirectly in a great variety of ways. The secretions from the mouth and nose doubtless contain the infection, and, while suspicion has not particularly fallen upon the feces and urine, it is probable that all the secretions and excretions from the body may be infective at some time throughout the disease, or during convalescence. Toys, pencils, books, letters, spoons, cups, towels, handkerchiefs, bedding, and objects of the greatest variety that have in any way come in contact with the patient may carry the infection. Under favorable circumstances the active principle may probably live for a considerable time upon fomites.

Smallpox is not usually considered an insect-borne disease, but it is highly probable that a fly lighting upon a smallpox patient and getting its proboscis, feet, and other portions of its body smeared with the variolous matter, and then flying to a susceptible person, could thus readily transmit the infection. Other insects may by such mechanical transfer play a similar rôle.

RESISTANCE OF THE VIRUS

It is generally, and doubtless correctly, assumed that the active principle of variola has approximately the same resistance to external conditions as vaccine virus. This assumption is confirmed by experimental evidence, which shows that the virus of smallpox is even more readily destroyed than the virus of cowpox. Scientific data concerning the viability of variolous matter is meager, owing to the fact that this question can only be settled by prolonged and repeated experiments upon monkeys. Brinckerhoff and Tyzzer[1] found that variolous virus is less resistant to desiccation than vaccine virus; that variolous virus does not pass a Berkefeld filter and is attenuated by long exposure to 60 per cent. glycerin.

In general it may be said that variolous virus is killed by exposure to ordinary germicidal substances, both liquid and gaseous, in the

[1] "Studies upon Experimental Variola and Vaccinia in Quadrumana," *Jour. Med. Research*, Vol. XIV, No. 2, Jan., 1906, pp. 223-359.

strengths and time commonly employed. It succumbs in fact before the average non-spore-bearing bacteria.

There is a probable exception to this statement in the case of carbolic acid and the coal-tar disinfectants. McClintock and Ferry [1] have shown that such germicides as carbolic acid, cresols, and the like do not destroy the virulence of vaccine virus in 0.5 per cent. solutions in five hours' exposure. In this strength and time almost all non-spore-bearing bacteria would be destroyed. The inference is allowable that this class of disinfectants cannot be relied upon to prevent the spread of smallpox.

SMALLPOX IN THE VACCINATED AND UNVACCINATED

The experience of over one hundred years offers convincing proof of the pronounced difference in the mortality and morbidity from smallpox in the vaccinated and the unvaccinated. The following table from Schamberg shows that, among thousands of cases of smallpox occurring in cities all over the world, the death rate from smallpox has been from five to sixteen times greater among the unvaccinated than among the vaccinated:

TABLE 2—DEATH-RATE FROM SMALLPOX AMONG VACCINATED AND UNVACCINATED IN VARIOUS COUNTRIES[2]

Places and Time of Observation	Total No. of Cases Observed	Death Rate per 100 Cases	
		Among the Unvaccinated	Among the Vaccinated
France, 1816-1841	16,397	16.125	1
Quebec, 1819-1820	?	27	1.66
Philadelphia, 1825	140	60	0
Canton Vaud, 1825-1829	5,838	24	2.16
Verona, 1828-1829	909	46.66	5.66
Milan, 1830-1851	10,240	38.33	7.66
Breslau, 1831-1833	220	53.8	2.11
Württemberg, 1831-1835	1,442	27.33	7.1
Carniola, 1834-1835	442	16.25	4.4
Vienna Hospital, 1834	360	51.25	12.5
Carinthia, 1834-1835	1,626	14.5	0.5
Adriatic, 1835	1,002	15.2	2.8
Lower Austria, 1835	2,287	25.8	11.5
Bohemia, 1835-1855	15,640	29.8	5.16
Galicia, 1836	1,059	23.5	5.14
Dalmatia, 1836	723	19.66	8.25
London Smallpox Hospital, 1836-1856	9,000	35	7
Vienna Hospital, 1837-1856	6,213	30	5
Kiel, 1852-1853	218	32	6
Württemberg (no date)	6,258	38.9	3½
Malta (no date)	7,570	21.07	4.2
Epidemiological Society Returns (no date)	4,624	23	2.9

[1] *Jour. of the Amer. Public Health Assn.*, June, 1911 (Vol. I, No. 6), p. 418.

[2] Extract from papers prepared in 1857 by Sir John Simon, Medical Officer of the General Board of Health of England, and at that time laid before Parliament with reference to the History and Practice of Vaccination. Published in first Report of the Royal Commission on Vaccination, 1889, Appendix 1, p. 74.

[1] *Jour. of the Amer. Public Health Assn.*, June, 1911 (Vol. 1, No. 6), p. 418.

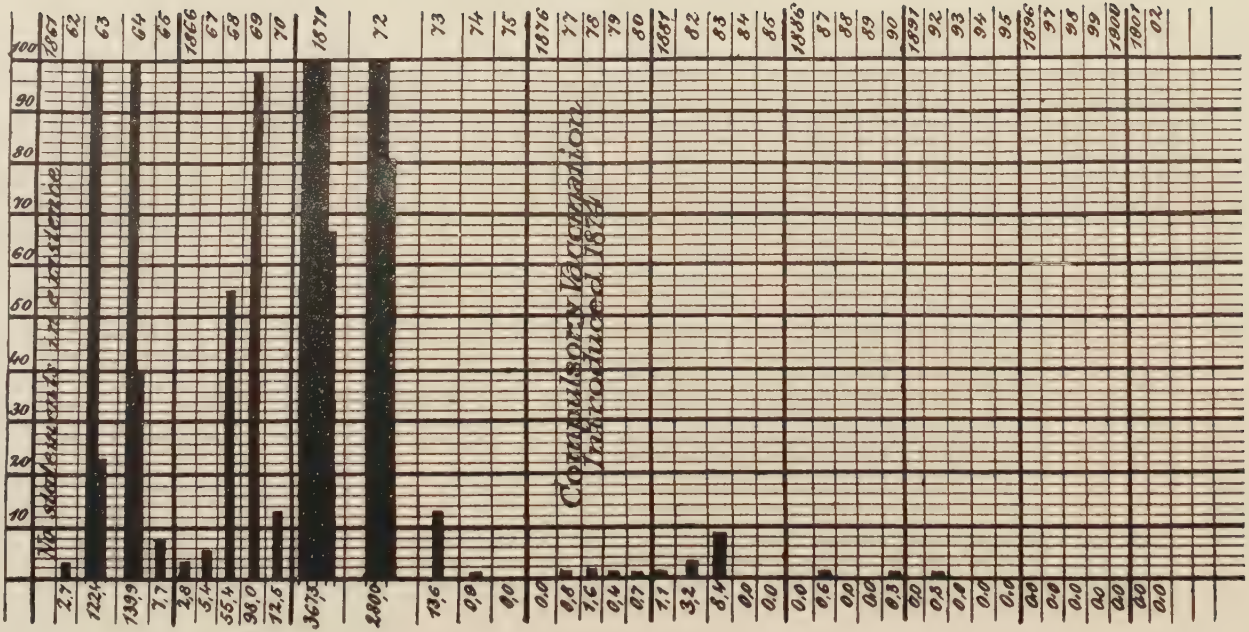


FIG. 5.—SMALLPOX MORTALITY PER 100,000 OF POPULATION IN BRESLAU. No compulsory vaccination before 1874; since then compulsory vaccination and revaccination (Schamberg).

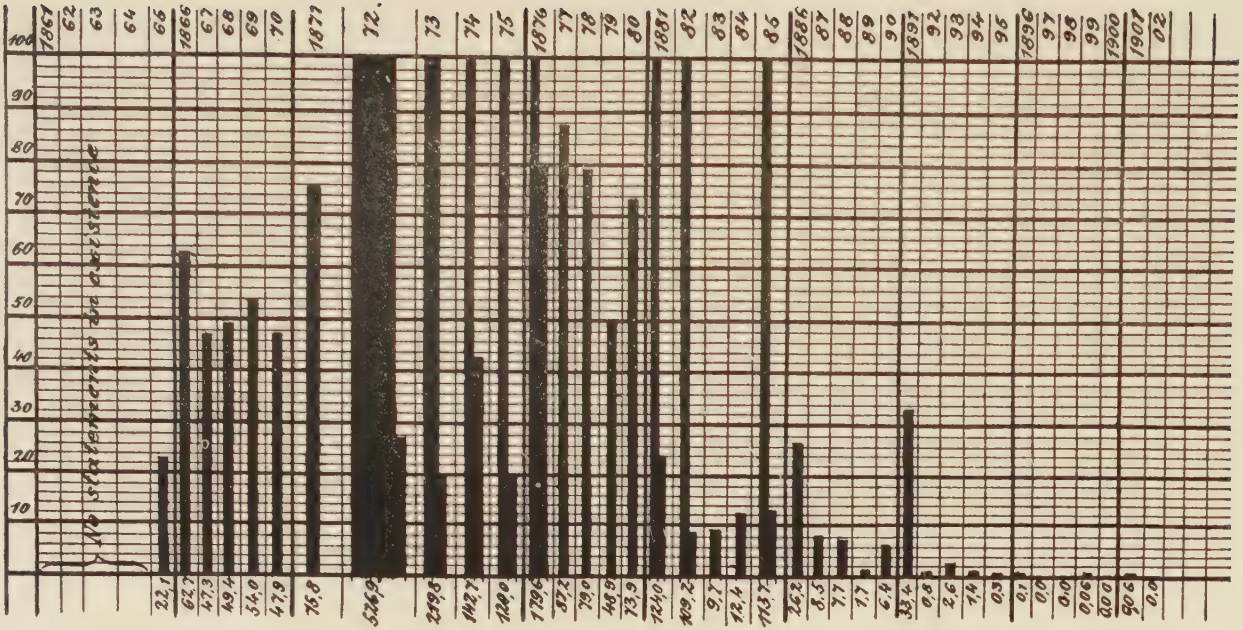


FIG. 6.—SMALLPOX MORTALITY PER 100,000 OF POPULATION IN VIENNA. No compulsory vaccination in Vienna, but since 1891 the administrative government authorities have used their best efforts in furthering vaccination (Schamberg).

TABLE 3—ANNUAL SMALLPOX DEATHS IN SWEDEN BEFORE AND AFTER THE INTRODUCTION OF VACCINATION[1]

Before Vaccination		After Vaccination	
1749[2]	4,453	1802	1,533
1750	6,180	1803	1,464
1751	5,546	1804	1,460
1752	10,302	1805	1,090
1753	8,000	1806	1,482
1754	6,862	1807	2,129
1755	4,705	1808	1,814
1756	7,858	1809	2,404
1757	10,241	1810[3]	824
1758	7,104	1811	689
1759	3,910	1812	404
1760	3,568	1813	547
1761	5,731	1814	308
1762	9,389	1815	472
1763	11,662	1816	690
1764	4,562		
1765	4,697	Compulsory Vaccination in Infancy	
1766	4,092		
1767	4,189	1817	242
1768	10,650	1818	305
1769	10,215	1819	161
1770	5,215	1820	143
1771	4,362	1821	37
1772	5,435		
1773	12,130	Total (20 years)	18,217
1774	2,065	1822	11
1775	1,275	1823	39
1776	1,503	1824	618
1777	1,943	1825	1,243
1778	6,607	1826	625
1779	15,102	1827	600
1780	3,374	1828	257
1781	1,485	1829	53
1782	2,482	1830	104
1783	3,915	1831	612
1784	12,456	1832	622
1785	5,077	1833	1,145
1786	671	1834	1,049
1787	1,771	1835	445
1788	5,462	1836	138
1789	6,764	1837	361
1790	5,893	1838	1,805
1791	3,101	1839	1,934
1792	1,939	1840	650
1793	2,103	1841	237
1794	3,964	1842	58
1795	6,740	1843	9
1796	4,503	1844	6
1797	1,733	1845	6
1798	1,357	1846	2
1799	3,756	1847	13
1800	12,032	1848	71
1801	6,057	1849	341
		1850	1,376
Total (53 years)	125,130	1851	2,488
		1852	1,534
		1853	279
		1854	204
		1855	41

[1] The population in 1751 was 1,785,727: in 1855 it was 3,639,332.
[2] From 1749 to 1773, inclusive, deaths from measles are included.
[3] First successful vaccination in Stockholm.

In countries like Germany, Sweden, Ireland, Scotland, and England, where vaccination is more or less compulsory, there is comparatively little smallpox. In countries like Belgium, Russia, Austria, and Spain, which have no compulsory vaccination laws, smallpox yearly claims many victims. See table 1, page 23.

THE RESULT OF VACCINATION IN GERMANY

April 8th, 1874, Germany passed a general compulsory vaccination and revaccination law. The law requires the vaccination of all infants before the expiration of the first year of life, and a second vaccination at the age of twelve. Since this law went into effect there have been no epidemics of smallpox in Germany, despite the fact that the disease has been frequently introduced from without. In 1897 there were but 8 deaths from smallpox in the entire German empire—population 54,000,000. Since then long periods have passed without a single death from smallpox. From 1901 to 1910 there were only 380 deaths from smallpox in Germany; during the same period there were 4,286 deaths from smallpox in England and Wales, with only about half the population of Germany; furthermore, many of the deaths in Germany were in foreigners: Thus in 1909, out of 26 deaths from smallpox, 13 were foreigners, 11 of whom were Russians. In the huge German army there have been only two deaths from smallpox since 1874. One of these was a reservist who had not been successfully vaccinated. Germany has taught the world how to utilize Jenner's great demonstration.

ISOLATION AND DISINFECTION

Isolation and disinfection are only secondary measures in preventing smallpox. They cannot be regarded as substitutes for vaccination.

Isolation should be carried out with strictness for the reason that smallpox is one of the most contagious of the communicable infections. While the patient should be isolated, it is not necessary to isolate the hospital by banishing it to an inconvenient or undesirable location. There is, in fact, no good reason why a smallpox hospital should not be one of the units of the general hospital for communicable diseases. In any event, there is no danger from a smallpox hospital situated upon a high road or near other habitations, provided always proper precautions are taken to prevent the spread of the disease.

The smallpox hospital should not be a pest house, but should be as inviting and attractive as economic conditions justify. Smallpox should not be treated in the home. From the standpoint of prophylaxis the hospital is the logical and best place to care for this and other communicable infections. If smallpox is treated in the home, this should

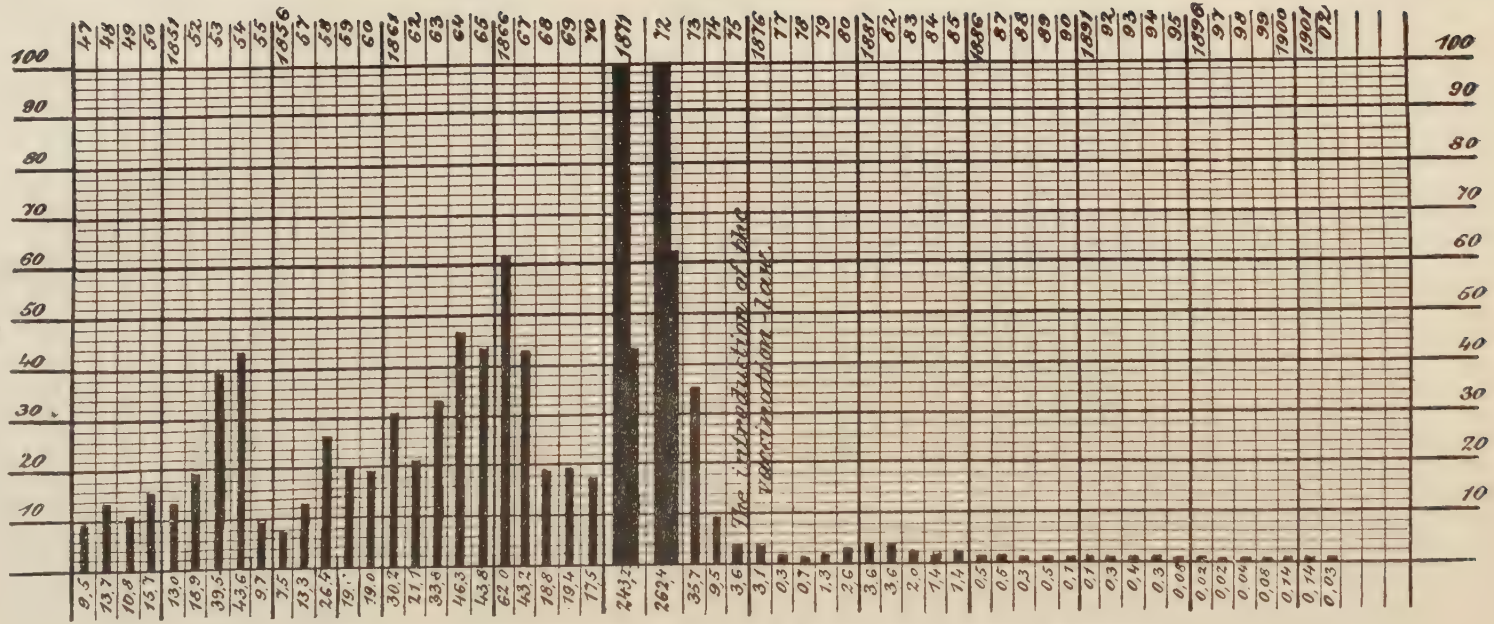


FIG. 7.—SMALLPOX MORTALITY PER 100,000 OF POPULATION IN PRUSSIA (Schamberg).

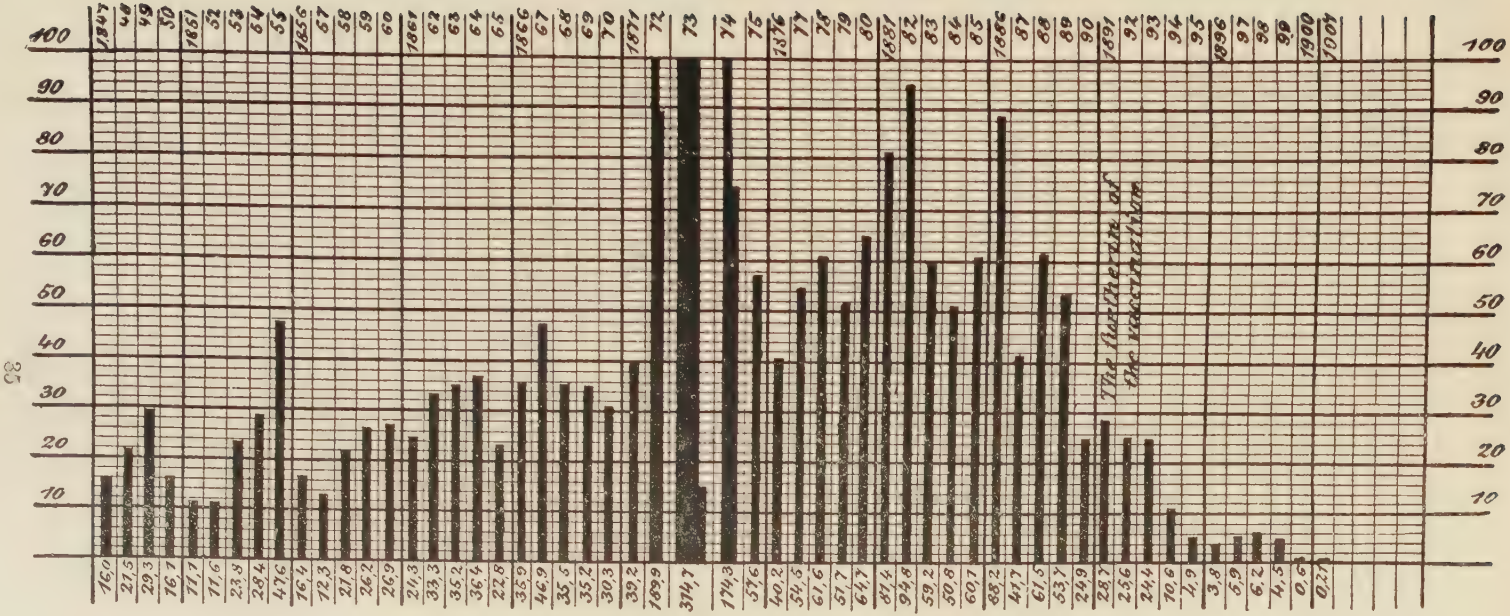


FIG. 8.—SMALLPOX MORTALITY PER 100,000 OF POPULATION IN AUSTRIA (Schamberg). There was very little difference in the number of deaths from the disease in Prussia and Austria as long as compulsory vaccination had not been introduced; since the enactment of the German vaccination law in Prussia, however, the mortality there has sunk to a previously unknown figure, whereas it has remained stationary and at the same high rate in Austria for many years. Up to 1889 the mortality from smallpox in the latter country was on an average greater than it was before the epidemic in 1872, and it is only since 1890 that favorable conditions have again prevailed, although the losses from smallpox have remained greater during recent years than in Prussia.

only be permitted if skilled nursing and trained attendants can be provided.

The room in which the smallpox patient is isolated should be simply furnished to facilitate cleanliness and to permit purification. It must be well screened and free from insects and vermin of all kinds. The room should be well ventilated. This may be accomplished by an open fireplace, in which case the contagium, if contained in the outgoing air, is burned in exit.

The nurse attending a case of smallpox should also be segregated, and all visiting should be strictly interdicted. A separate kitchen should be provided and care should be taken that the dishes be scalded and remnants of food burned.

Bedding, underwear, towels, and other objects should not leave the sick room unless they are first boiled, steamed, or immersed in a suitable germicidal solution, such as bichlorid of mercury, 1-1,000, or formalin, 10 per cent. Carbolic acid should not be trusted.

For terminal disinfection either sulphur dioxid or formaldehyde may be used. Objects particularly contaminated or soon to be used by others should be given a separate and special disinfection. Finally, the room should be thoroughly cleansed, aired, and sunned.

The patient must be regarded as the source and fountainhead of the infection, and measures should be used at the bedside to prevent the surroundings from becoming contaminated. Cloths, cotton, and other dressings that become soiled with the contents of the vesicles and pustules after they break should be burned. The urine and feces may be disinfected with chlorinated lime. The sputum and discharges from abscesses must be carefully disinfected by an approved method (see Section XII). As a rule, smallpox patients are not dismissed from quarantine until desquamation has ceased. This may be favored by the use of warm baths and a generous use of soap, also by anointing the skin with vaselin or a bland oil. Special attention should be given to the hair, which should be well shampooed, to the interdigital spaces, and the fingernails, as well as to all folds of the skin, before the patient is released.

The management of smallpox epidemics is discussed on page 319.

RABIES

Synonyms.—*Hydrophobia; Wasserscheu, Wuth, Tollwuth* (German); *Lyssa* (Greek); *La Rage* (French).

Rabies is an acute, specific, rapidly fatal infection communicated from a rabid animal to a susceptible animal, usually through a wound

[1] Collateral reading: "Facts and Problems of Rabies," Stimson, *Hyg. Lab. Bull. No. 65,* U. S. P. H. & M. H. S.

produced by biting. Man always contracts the disease from some lower animal, usually the dog. The infective agent must be inoculated into the tissues; the virus is harmless when ingested. Rabies may be regarded as a wound infection. The specific principle is contained in the saliva of animals suffering with the disease. The infection, therefore, may be conveyed by licking provided there are fissures or open wounds in the skin. It is also possible to introduce the virus through autopsy accidents and other unusual ways, but commonly it is introduced through wounds produced by the teeth of a rabid animal.

Every mammalian animal is susceptible. Even birds may contract the disease. It is most common in dogs, but it also occurs frequently in wolves, jackals, foxes, and hyenas. Rabies in cats is comparatively rare. Cattle, sheep, and goats are infected relatively in about the same degree. It is less common in horses. Swine contract the disease less frequently than other domestic animals. Skunks have the disease and sometimes transmit it to man.

Although all mammals are susceptible to rabies, it is perpetuated in nature almost exclusively by the domestic dog, only to a small extent by wild animals of the dog family, and occasionally by skunks, cats, etc. Outbreaks have been reported under unusual circumstances. Thus Carini [1] reports an epizootic causing the death of about 4,000 cattle and 1,000 horses in Sao Paulo, Brazil. There was no unusual prevalence of rabies in dogs at the time but it was noticed that bats, in broad daylight, attacked and bit the cattle, and Carini suggests that bats may have been the source of the extensive epizootic. The animals affected all died after a few days and the meat and hides were utilized but no mishaps have been known to follow.

Rabies exists practically all over the world, except in Australia, and recently in England. It is most common in France, Beligum, and Russia. In the United States 111 human deaths were reported in 1908. In the same year there were 535 localities in which rabid animals were reported; in 1911 there were 1,381 localities, and 98 deaths in man. In 1890 the United States census reported 143 deaths in 30 states, and in 1900 but 23 deaths.

Rabies is remarkable in that the mortality is 100 per cent. After symptoms begin recovery never occurs in man or other animals. Joseph Koch (1910), however, describes an abortive rabies. The disease is peculiar in several other particulars, especially the period of incubation, which is more variable and more prolonged than that of any other acute infection.

Rabies is commonly supposed to prevail only during the hot months, but it may be just as bad in cold weather. In fact, exposure to cold seems to increase its virulence. More cases occur from April to Sep-

[1] *Ann. de l'Inst. Pasteur,* Paris, Nov., XXV, 11, p. 785.

tember than from October to March in this climate, because dogs run abroad more freely at this season of the year. It is this fact, and not the temperature, that influences the prevalence of the disease.

Period of Incubation.—From the standpoint of prevention it is fortunate that the period of incubation of this disease is prolonged. This period varies from 14 days to a year or more. The average period is as follows: Man, 40 days; dogs, 21-40 days; horses, 28-56 days; cows, 28-56 days; pigs, 14-21 days; goats and sheep, 21-28 days; birds, 14-40 days.

The period of incubation depends largely upon the site of the wound, the relation to the nerve, the amount and virulence of the virus. It requires about 15 days to induce an active immunity to the disease by means of the Pasteur preventive treatment. There is, therefore, usually plenty of time, if the case is seen early, to prevent the development of symptoms.

It is probable that the prolonged period of incubation is due in part to the fact that the living principle reaches the central nervous system, but remains dormant until favorable conditions permit multiplication and the production of toxic effects (Joseph Koch).

Entrance and Exit of the Virus.—The active principle of rabies occurs principally in the saliva and in the central nervous system. It may be in the saliva at least three days (possibly eight) before the animal shows symptoms (Roux and Nocard). It is, therefore, sufficient to watch a dog that has bitten a person or another animal for ten days. If no symptoms of rabies appear during this time there is no danger of conveying the disease, and the Pasteur treatment is unnecessary.

The virus may also be found in the adrenals, the tear glands, the vitreous humor, the spermatic fluid, the urine, the lymph, the milk, as well as all parts of the central nervous system and the peripheral nerves. It is also found in the spinal and ventricular fluids. It has not been demonstrated in the liver, spleen, blood, or muscles.

The virus enters the system through the broken skin and follows the nerve trunks from the seat of injury to the spinal cord, thence to the medulla and brain. The route corresponds to that of tetanus toxin. The mode of invasion of the virus may explain why pain, throbbing, tingling, numbness and other nervous disturbances are the first symptoms to occur in parts of the body that have received the virus. It also partly explains the variable period of incubation, which is shorter in wounds of the face than in wounds of the extremities. It also explains why the disease is more serious when the wounds are in parts of the body where there is an abundant nerve supply.

The Relative Danger of Bites.—Wolf bites are most dangerous on account of the savage character of the wound, and the virulence of the virus. Cat bites come next, and then dog bites. The relative danger

of bites of other animals is as follows: foxes, jackals, horses, asses, cattle, sheep, pigs. There is no authentic instance of the transmission of the disease by the bite of man, though this may be possible. The bites of horses and other herbivora are less dangerous because their blunt teeth usually cause contused wounds without breaking the skin.

Bites on exposed surfaces are more dangerous than through the clothing, because the saliva is wiped from the teeth and little or none enters the wound. Long-haired dogs and sheep often escape infection for the same reason. Bites upon the face are most apt to be followed by rabies.

Not every person bitten by a mad animal develops rabies. Leblanc's figures are 16.6 per cent. The statistics are difficult to analyze, and it is almost impossible now to collect sufficient data. According to the most reliable data, it would seem that rabies develops in not less than one person in ten bitten by mad dogs, and not receiving the Pasteur treatment. Paltauf places the figures at 6 to 9 per cent.

Viability.—The virus of rabies in the spinal cord of rabbits dies in about 14 days when dried at 20°-22° C., if protected from putrefaction and light. Spread in thin layers, it dies in 4 or 5 days, and exposed to the sunlight in 40 hours. It is quite resistant to putrefaction. In a decomposed carcass it may be recovered by placing some of the central nervous system in glycerin. The glycerin destroys most of the contaminating bacteria, but preserves the virus. Rabies virus is completely destroyed at 50° C. in one hour, and at 60° C. in 30 minutes. It is not injured by extreme cold. Five per cent. carbolic acid for one hour, 1-1,000 bichlorid of mercury for one hour, or a saturated solution of iodin in water completely destroys its virulence.

PROPHYLAXIS

The prevention of rabies is considered under three heads: (1) Treatment of the wounds; (2) the Pasteur prophylactic treatment, and (3) the control of the disease in dogs by muzzling and quarantine.

The cauterization of the wound and the Pasteur prophylactic treatment are efficient preventive measures for the individual, but they are not the true and best methods of controlling and preventing rabies. The disease may be avoided, even exterminated, by an intelligent system of muzzling and quarantining of dogs. A high tax on dogs and leashing are only restrictive measures. In England, when the dogs were muzzled, rabies diminished. The law was repealed, owing to misplaced sympathy for the dog, and rabies promptly increased. The law was again enforced, and in about two years the disease disappeared (see the accompanying chart). Now a strict quarantine of six months is maintained against dogs entering England. It is no longer necessary to

muzzle dogs in England, but muzzles will again be required should the disease reappear. Consistent muzzling of all dogs for two years will practically exterminate rabies. In Australia there are few carnivorous animals, mostly marsupials; there rabies does not exist, for it has been kept out owing to early and effective quarantine measures.

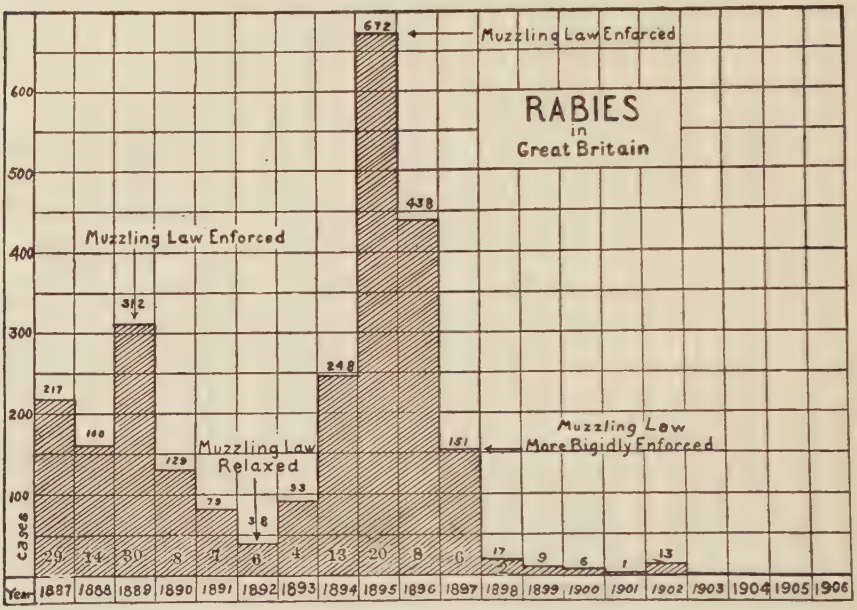


FIG. 9.—CHART SHOWING RELATION OF ENFORCEMENT OF MUZZLING LAW TO PREVALENCE OF RABIES IN GREAT BRITAIN. The figures in the cross-hatching indicate the number of persons who died of rabies in England and Wales. The ordinates represent cases in dogs. (Frothingham.)

Prophylactic measures necessary to control the dog question are: the destruction of ownerless dogs; license fee and tag for all dogs; owners to be legally responsible for damage inflicted by their dogs; education of the dog-owning public concerning the spread of communicable diseases, especially rabies; compulsory reporting of all cases or suspected cases of rabies. Further special and temporary measures advocated are: muzzling; restraint with chains, leash, etc.; observation in quarantine, or killing of all animals bitten by dogs; disinfection, etc.

THE LOCAL TREATMENT OF THE WOUND

Wounds produced by the bite of an animal in which there is any suspicion of rabies should at once be cauterized with fuming nitric acid. The acid is best applied with a glass rod very thoroughly to all the parts of the wound, care being taken that pockets and recesses do not escape. Thorough cauterization at once reduces the danger of wound

complications, and experience demonstrates that wounds so treated at once, are practically never followed by rabies. Marie obtained conflicting results with local treatment in experimental rabies; Cabot obtained the best results in a series of extensive experiments with nitric acid. Poor [1] was able to save the lives of 45 per cent. of guinea-pigs by cauterization with nitric acid at the end of 24 hours. In the absence of nitric acid the actual cautery may be used. Strong antiseptics, such as carbolic acid and formalin, are less reliable. Nitrate of silver is valueless. In any wound produced by the bite of an animal the rule is to cauterize unless sure that the animal is not mad.

It has been shown that the virus may remain alive and virulent in the scar for a long time, and it has become a question whether patients seen after the wound has healed should not have the scar excised; this, however, is not the present practice.

THE PASTEUR PROPHYLACTIC TREATMENT

This method of prophylaxis was announced December 6, 1883, by Pasteur, at the International Congress at Copenhagen, and on February 24, 1884, he laid before the French Academy the details of his experiments and results. The next year Pasteur, with the help of Roux and Chamberland, worked out the details of the method now used.

The principle of the treatment consists in producing an active immunity by means of an attenuated virus. The virus is attenuated by drying. The fixed virus contained in the spinal cord of rabbits dead of hydrophobia is the material used, for subcutaneous injection.

Street Virus and Fixed Virus.—The distinction between fixed and street virus is of fundamental importance in reference to the question of immunity. Street virus refers to the virus obtained from mad dogs naturally infected. When this virus is inoculated into a rabbit, it reproduces the disease after a period of incubation of from 14 to 21 days or more. This street virus may then be conveyed from rabbit to rabbit through a number of transfers. In the passage from rabbit to rabbit the virus becomes more virulent for rabbits, but less so for dogs and other animals. The period of incubation is progressively shortened, until finally the rabbits invariably sicken on the sixth or seventh day and die on the ninth or tenth. When the virus has reached this degree of virulence for rabbits, it is said to be "fixed," for the reason that its potency remains constant. In its passage through rabbits the modification from street virus to fixed virus is gradual. It is important to note that fixed virus, which has attained a high degree of virulence for rab-

[1] *Collected Studies,* Research Lab., Dept. of Health, City of N. Y., VI, 1911, p. 25.

bits, has lost much of its virulence for dogs, and is probably entirely avirulent for man.

Proescher[1] injected into himself the entire brain and medulla of a rabbit (fixed virus), and another entire brain into a volunteer. No ill effects of any kind were noted in either case. A control rabbit injected with a 0.02 dilution of the same emulsion died in seven days with experimental rabies.

Marx tested the fresh fixed virus upon monkeys in large doses, with negative results. Ferran in Barcelona in 1887 inoculated 85 persons with the fresh fixed virus as a prophylactic treatment for dog bites with good results, which have been further confirmed by Wysokowiez and Nitsch. The evidence points clearly to the fact that the fixed virus of rabbits does not produce rabies in man when introduced into the subcutaneous tissue.

Preparation of the Virus.—Rabbits are injected under the dura mater with a few drops of an emulsion of fresh fixed virus obtained from the pons or medulla of another rabbit dead of hydrophobia. Strict aseptic precautions are necessary in order to keep out other infections. The rabbit should begin to show symptoms on the sixth or seventh day, and die on the ninth or tenth. Usually the rabbit is not allowed to die, but is chloroformed on the last day in order to avoid terminal infections and unnecessary suffering. The spinal cord is removed and hung in a bottle containing potassium hydroxid. These bottles are kept in the dark at a temperature of 20°-22° C. Under these conditions the cord gradually desiccates, and at the same time the virulence of the virus diminishes, until the fourteenth day, when it is no longer infective. This is why Pasteur started the treatment with a cord fourteen days old.

One half a cubic centimeter of the cord constitutes a dose. This is ground in sterile salt solution so as to produce a uniform emulsion, which is injected into the subcutaneous tissue of the abdominal wall. In many institutes the small segments cut each day from the drying cord are placed in pure glycerin. The virulence of the cord in glycerin is not altered for at least 30 days, if kept in the dark and at 15° C. This method, introduced by Calmette, is very convenient, especially where comparatively few patients are treated. Glycerin has the added advantage of destroying infections due to non-spore-bearing bacteria that may be present. As a further precaution, bacteriological examinations are made of parts of the spinal cord in order to insure the absence of bacteria, and the rabbit is carefully autopsied as a guarantee that no other disease is present.

The scheme of treatment advocated by Pasteur and still used at l'Institut Pasteur in Paris and many other places is as follows:

[1] *N. Y. Med. Jour.*, Oct. 9, 1909, also *Arch. of Int. Med.*, Sept., 1911, VIII, 3, p. 353.

PASTEUR PROPHYLACTIC TREATMENT—RECOMMENDED BY PASTEUR

Mild Treatment			Intensive Treatment		
Day of Treatment	Age of the Dried Cord	Amount of Injected Emulsion 1 cm. to 5 c. c.	Day of Treatment	Age of the Dried Cord	Amount of Injected Emulsion 1 cm. to 5 c. c.
1	14 Days	3 c. c.	1	14 Days	3 c. c.
	13	3		13	3
				12	3
				11	3
2	12	3		10	3
	11	3	2	9	3
				8	3
				7	3
3	10	3	3	6	2
	9	3		6	2
4	8	3	4	5	2
	7	3			
5	6	2	5	5	2
	6	2			
6	5	2	6	4	2
7	5	2	7	3	1
8	4	2	8	4	2
9	3	1	9	3	1
10	5	2	10	5	2
11	5	2	11	5	2
12	4	2	12	4	2
13	4	2	13	4	2
14	3	2	14	3	2
15	3	2	15	3	2
16	5	2	16	5	2
17	4	2	17	4	2
18	3	2	18	3	2
			19	5	2
			20	4	2
			21	3	2

Many Pasteur institutes now use a modified treatment, starting with an 8-day instead of a 14-day-old cord, which is exemplified in the scheme on next page, used at the Hygienic Laboratory, Public Health Service.

The Pasteur scheme has been further modified in various ways. Bujwid and Babes use stronger treatment than that advocated by Pasteur. Puscariu in Jessy uses a method based upon the experiments of Babes, which show that an emulsion of fixed virus when heated to 50°-58° C. is attenuated in virulence. Tizzoni and Cattani attenuate the virus in gastric juice, and Höyges simply dilutes the fresh virus. The original dilution is 1-100, and the first dose is one ten-thousandth of this. Ferran in Barcelona, Proescher in Pittsburgh, and others inject patients with the unaltered, fresh, fixed virus. The advantages of using the virus as fresh and strong as possible are that an active immunity is produced more quickly, and this is of considerable importance in wounds of the face; also in wolf and cat bites, which frequently have a short period of incubation. Further, only one or two injections

PASTEUR PROPHYLACTIC TREATMENT—HYGIENIC LABORATORY, WASHINGTON, D. C.

Day	Age of the Dried Cord	Amount		
		Adult	5 to 10 Years	1 to 5 Years
	Scheme for Mild Treatment			
1	8–7–6	2.5 c. c.	2.5 c. c.	2.0 c. c.
2	5–4	2.5	2.5	1.5
3	4–3	2.5	2.5	2.0
4	5	2.5	2.5	2.5
5	4	2.5	2.5	2.5
6	3	2.5	2.5	2.0
7	3	2.5	2.5	2.0
8	2	2.5	1.5	1.0
9	2	2.5	2.0	1.5
10	5	2.5	2.5	2.5
11	5	2.5	2.5	2.5
12	4	2.5	2.5	2.5
13	4	2.5	2.5	2.5
14	3	2.5	2.5	2.0
15	3	2.5	2.5	2.0
16	2	2.5	2.0	1.5
17	2	2.5	2.0	1.5
18	4	2.5	2.5	2.5
19	3	2.5	2.5	2.5
20	2	2.5	2.5	2.0
21	2	2.5	2.5	2.0
	Scheme for Intensive Treatment			
1	8–7–6	2.5 c.c.	2.5 c.c.	2.5 c.c.
2	4–3	2.5	2.5	2.0
3	5–4	2.5	2.5	2.5
4	3	2.5	2.5	2.0
5	3	2.5	2.5	2.0
6	2	2.5	2.0	1.5
7	2	2.5	2.5	2.0
8	1	2.5	1.5	1.0
9	5	2.5	2.5	2.5
10	4	2.5	2.5	2.5
11	4	2.5	2.5	2.5
12	3	2.5	2.5	2.0
13	3	2.5	2.5	2.0
14	2	2.5	2.5	2.0
15	2	2.5	2.5	2.0
16	4	2.5	2.5	2.5
17	3	2.5	2.5	2.5
18	2	2.5	2.5	2.0
19	2	2.5	2.5	2.0
20	3	2.5	2.5	2.5
21	2	2.5	2.5	2.0

of the fresh virus are necessary to produce an immunity, and this shortens and simplifies the treatment very much.

Harris [1] has shown that rabic material may be completely desiccated without destruction of virulence, provided the dehydration takes place at a low temperature. The lower the temperature the greater will be the amount of virulence preserved. Virus so desiccated contains per weight as much infectivity as the fresh virus. The loss of virulence of the dried virus is so slow that it may be standardized, permitting an accuracy of dosage hitherto impossible. The unit is the smallest amount which, when injected intracerebrally into a full-grown rabbit, will produce paresis on the seventh day. The use of this desiccated virus in the prophylactic immunization of animals and persons offers many advantages over other methods.

Treatment at a distance from a Pasteur institute is now possible by sending a piece of cord, or the emulsion in glycerin.

Care During the Treatment.—During the treatment the patient may go about his usual business. It is not necessary to stay in bed. The patient should, however, avoid fatigue, cold, and alcohol. It has been shown that these are important predisposing factors to the disease. It was found that customs' officers returning to the Siberian borders after prophylactic treatment for wolf bites showed an unusual mortality, which seemed to be due to exposure to cold. The disease has been observed to be brought on after a cold bath, falling into the water, and similar depressing influences.

Complications of the Treatment.—The Pasteur prophylactic treatment may be complicated by (1) local reactions or (2) paralysis.

Local reactions at the site of the wound are usually trivial. Abscesses almost never occur. The local reactions consist of redness and induration. Their occurrence increases with the progress of the treatment; they are most frequent in the second week. As the treatment involves the introduction of a large quantity of foreign proteins into the body, it is probable that these reactions represent a phase of hypersusceptibility. (See Anaphylaxis.)

Paralysis.—Paralysis occasionally occurs and may be fatal. There is doubt concerning the cause of this paralysis, and a question whether it may be a mild or modified type of rabies, or a form of anaphylaxis. In a case treated at the Hygienic Laboratory the paralysis came on 18 days after treatment, and was transient. The New York Pasteur Institute reports a death from "ascending paralysis," which came on four days after the treatment. W. A. Jones [2] reported two cases with recovery. In 1905 Remlinger, head of the Constantinople Institute for Rabies, reported 40 cases of paralysis; Müller found 16 cases in the literature, and had two of his own; Panpoukis, three cases; Jones, 2; making a total of 63, 2 of whom died.

[1] *Jour. of Infect. Dis.*, May, 1912, X, 3, pp. 369-377.
[2] *Jour. A. M. A.*, Nov. 13, 1909, p. 1626.

The Immunity.—Duration.—The immunity appears two weeks after the treatment and lasts a varying period of time, depending upon the individual—at least for several years. In this respect it does not differ from other instances of acquired immunity. The fact that the immunity appears on about the fifteenth day after the end of the treatment was discovered by Pasteur as a result of animal experimentation. The statistics of the Pasteur Institute, giving the mortality from rabies in persons following the prophylactic treatment, exclude instances in which the disease develops within fifteen days after the end of the treatment.

Nature.—The nature of the immunity is not clear. It certainly is not due to an antitoxin. Immune bodies are demonstrable in the blood twenty days after the last injection. This is determined by mixing *in vitro* the active virus with the blood serum, which neutralizes its activity. This neutralization is generally considered to be microbicidal or lytic in nature.

Degree.—The degree of the immunity also varies, as is evidenced by the fact that a certain small percentage of the persons treated die of rabies.

The Results of the Treatment.—Statistics giving the results of the treatment are somewhat difficult to analyze, as many factors are unobtainable. Patients should be kept under observation at least a year. Exceptional cases occur one year following the treatment. Cases that occur within fifteen days after the treatment are excluded from the French statistics, for reasons that have already been stated. The figures on this basis show a mortality which averages about 0.5 per cent. Better results are being obtained from year to year.

The table on the following page gives the general results at l'Institut Pasteur, Paris, since beginning the treatment.

When we compare these figures with the fact that from 6 to 10 per cent. and sometimes 16.6 per cent. of all persons bitten by rabid dogs die of rabies, the prophylactic value of the Pasteur treatment is evident.

Some series of cases give a much higher mortality. Thus, of 855 cases collected by Tordieu, Thamehayn, and Bouley, 399 ended in death, or 46.6 per cent. In another series of cases given by Bouley, out of 266 persons bitten by mad dogs, 152 died of hydrophobia. But of these 120 were bitten on the face and hands, the greater danger of which has been mentioned. The mortality of bites from wolves is placed at from 60 to 80 per cent.

Contraindications.—There are no particular contraindications to the treatment. All ages and conditions should be treated if exposed. Apparently no harm is done pregnant women. I have injected patients having malaria without trouble following. The treatment may be con-

RESULTS OF TREATMENT AT L'INSTITUT PASTEUR, PARIS.

Year	Persons	Deaths	Mortality
1886	2,671	25	0.94%
1887	1,770	14	0.79
1888	1,622	9	0.55
1889	1,830	7	0.38
1890	1,540	5	0.32
1891	1,559	4	0.25
1892	1,790	4	0.22
1893	1,648	6	0.36
1894	1,387	7	0.50
1895	1,520	5	0.38
1896	1,308	4	0.30
1897	1,521	6	0.39
1898	1,465	3	0.20
1899	1,614	4	0.25
1900	1,420	4	0.28
1901	1,321	5	0.38
1902	1,005	2	0.18
1903	628	2	0.32
1904	755	3	0.39
1905	727	3	0.41
1906	772	1	0.13
1907	786	3	0.38
1908	524	1	0.19
1909	467	1	0.21

tinued in patients having colds, fevers, and other ailments without noticeable harm.

When to Give the Pasteur Treatment.—It is sometimes difficult to decide whether the Pasteur prophylactic treatment should or should not be given. The treatment causes sufficient personal inconvenience, not to speak of the danger (however slight) of paralysis, to avoid advising it if unnecessary. In many cases it is impossible to discover whether the dog which inflicted the bite is mad or not. The rule in cases of doubtful exposure is to advise the treatment.

Persons not infrequently apply for advice giving the following history: They have not been bitten, but they have been licked on the hands and face by a dog which subsequently developed the disease. Persons are sometimes similarly exposed by washing the mouth of a rabid horse. In these cases the important question is whether there were fissures or abrasions in the skin at the time. There may be little wounds in the skin not evident to the naked eye. In such cases the danger is slight, but in apprehensive subjects the assurance of protection which the treatment affords is an important element in arriving at a decision.

In all cases it is important to know whether the dog is mad or not. If the dog can be found and kept under observation for 10 days and no symptoms appear, the Pasteur treatment is not necessary. Animals killed early in the course of rabies may fail to show the microscopic

evidence of the disease, thus causing an indefinite delay in diagnosis awaiting inoculation tests. Should symptoms develop, the question of diagnosis is all-important.

Diagnosis of Rabies in Dogs.—The diagnosis of rabies in dogs may be made in three ways: (1) from the symptoms; (2) from the presence of Negri bodies in the central nervous system, and (3) by animal inoculations.

1. The symptoms may be very suggestive, but a diagnosis must always rest upon the pathological lesions and the inoculation tests. The course of the disease may be divided into three stages: a premonitory stage, a stage of excitement, and a paralytic stage. The first two stages may be absent or transient. All rabid animals invariably become paralyzed before they die. In dogs the first symptom consists solely in a change in the disposition of the animal. He is easily excited, but does not show a disposition to bite. Soon the restlessness becomes more marked, and the animal may become furious and even show signs of delirium. The animal does not fear water, as is commonly supposed, but rushes about attacking every object in his way. Dogs suffering from furious rabies have a tendency to run long distances (25 miles or more) often biting and inoculating large numbers of other animals and persons en route. Very soon paralysis sets in, commencing in the hind legs, and finally becomes general. The course of the disease is always rapid, averaging from 4 to 5 days, rarely exceeding 10 days. When the stage of excitement is brief or absent, the disease is known as dumb rabies. This is the prevailing type in Turkey. This explains the relative rarity of rabies in man in Turkey, where dogs abound.

2. There is a difference of opinion concerning the significance of the Negri bodies (*Neuroryctes hydrophobiæ*), which, however, are very constant in rabies and peculiar to it. If Negri bodies are found in the dog, the Pasteur treatment should be started at once. The absence of Negri bodies, however, does not necessarily mean the absence of rabies. These bodies are sometimes difficult to find, or may not be present in the parts of the central nervous system which are examined. Negri bodies for diagnostic purposes may best be demonstrated by impression preparations stained according to Van Gieson, as recommended by L. Frothingham; or smears stained by the Mallory eosin-methylene-blue method recommended by Williams and Lowden. Smears are prepared by crushing a small portion of the brain matter between two slides; portions are selected from Ammon's horn and also from the cerebellum, cerebral cortex, and medulla. These smears are then fixed and stained as follows:

(a) Zenker's solution for 15 minutes.

(b) Wash in tap water.

(c) Ninety-five per cent. alcohol tinted with iodin.

(d) Absolute alcohol five minutes.

(e) Five to ten per cent. watery solution of eosin (Grübler W. g.) five minutes.

(f) Stain in Unna's polychrome methylene blue two to three minutes.

(g) Wash in water.

(h) Differentiate in ninety-five per cent. alcohol.

(i) Blot off, dry, and examine with oil immersion lens.

The lesions of Van Gehuchten and Nélis, described in 1900, are the most characteristic anatomical changes. These lesions are found in the peripheral ganglia of the cerebrospinal and sympathetic systems, especially in the plexiform ganglia of the pneumogastric nerve, and also the Gasserian ganglia. The normal nerve cells of these ganglia lie in a capsule lined with a single layer of endothelial cells. In rabies these endothelial cells proliferate and the nerve cells are pushed aside and even destroyed. The ganglion may finally contain only round cells.

3. The final diagnosis of rabies rests upon animal experimentation. A small quantity of the suspected material is placed under the dura mater of a rabbit or guinea-pig. The diagnosis by this method, however, requires so much time (on account of the long period of incubation of the disease) that it is of no practical value in deciding whether or not the Pasteur prophylactic treatment should be given, but in any critical case the positive evidence furnished by animal experimentation is incontrovertible.

THE VENEREAL DISEASES

As a danger to the public health, as a peril to the family, and as a menace to the vitality, health, and physical progress of the race, the venereal diseases are justly regarded as the greatest of modern plagues, and their prophylaxis the most pressing problem of preventive medicine that confronts us at the present day.

There are three venereal diseases: syphilis, gonorrhea, and chancroid. In order to have a clear understanding of the problems of venereal prophylaxis it is necessary to have a knowledge of the essential features of these preventable infections. Two of them, syphilis and gonorrhea, are of great importance, because they are very prevalent and because they are very serious infections with grave consequences.

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SYPHILIS

There are many striking things about syphilis, but nothing so striking as its persistence in spite of knowledge complete enough to stamp it out and in view of the popular dread in which the disease is held. It is preventable, even curable—yet scarcely another disease equals it in the extent and intensity of its ravages.

Syphilis is a good illustration of the fact that it is much more difficult to control a disease transmitted directly from man to man than a disease transmitted by an intermediate host, or one in which the infective principle is transferred through our environment. We have a certain amount of control over our surroundings, and we have dominion over the lower animals, but the control of man requires the consent of the governed.

Civilization and syphilization have been close companions, but syphilis is now less prevalent among civilized than uncivilized peoples—this is promising. Civilization, however, should not be content until it has controlled syphilis as effectively as it has a few other preventable infections. The effort to do so, at least, must be persistent and sincere.

From the economic side, syphilis is not a serious disease in its primary and secondary stages; that is, persons with syphilis during the early stages are usually not ill enough to cease work. Acutely fatal cases, such as frequently occurred in the sixteenth century, are now rare; in other words, the disease has lost much of its early virulence. It is the late manifestations, the sequelæ and the so-called parasyphilitic lesions, as well as the inherited consequences of the disease, that cause great economic loss. About one-fifth of all the insane in our asylums are cases of general paresis; 90 per cent. of these give the Wassermann reaction. Syphilis, alcohol, and heredity fill our insane asylums.

The consequences of syphilis are often more severe upon the offspring than upon the syphilitic parent. The infection itself, or various defects, especially of the nervous system, resulting from the consequences of syphilis, may be transmitted from parent to child, often with fatal results. When death does not ensue the results may be still more tragic.

Syphilis is an infection caused by the *Treponema pallidum* (formerly known as the *Spirochæta pallida*). It is a communicable disease acquired by direct contact with infected persons or things. It runs a chronic course with local and general manifestations, usually divided into three stages, which are not always well defined. The primary stage consists of the chancre which forms at the site of the initial infection. The chancre is a hard indurated ulcer in the skin or mucous membrane, and appears about three weeks (not less than ten days)

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after the receipt of the infection. The secondary stage is characterized by a general invasion of the spirochete throughout the system, as indicated by a general involvement of the lymph nodes, eruptions upon the skin and mucous membranes, fever, anemia, and other indications of a generalized infection. The third stage is characterized by a localized granulomatous growth known as a gumma. Gummata may appear in almost any tissue or organ of the body. A fourth stage is sometimes added to the picture, consisting of the sequelæ or parasyphilitic phenomena, such as general paresis, arteriosclerosis, locomotor ataxia, aneurysm, etc.

The health officer should regard syphilis just as he does the acute febrile exanthematous diseases. Because syphilis runs a slow and often chronic course with mild constitutional symptoms during its early stages, it is often placed in a class by itself. This is a mistake. Syphilis has its period of incubation, eruption, and decline, just as measles and smallpox have.

There is no natural immunity to syphilis; all are susceptible, but the severity of individual cases varies greatly. This is due either to the virulence of the strain, the amount of the infection, or to variation in individual resistance. The disease is now much less severe than it was following the pandemic which spread over the civilized world after 1494, when the army of Charles VIII, 32,000 strong, started out to conquer the Italian peninsula.

One attack of syphilis confers an immunity, in that reinfections do not produce another chancre. That is, the virus cannot be inoculated upon a person who has or has had the disease. The immunity is peculiar in that, while the person cannot have a second chancre, this fact has no influence upon the development of the secondary and tertiary lesions resulting from the first infection. For Colles' and Profeta's laws of syphilitic immunity and the transmission of syphilis see page 447.

In a large majority of all cases of syphilis the infection is transmitted during sexual approach. It is, therefore, spoken of as a venereal disease; many cases, however, are contracted out of venery. These accidental infections are more common than is ordinarily supposed. Metchnikoff reports that a great number of cases of non-venereal syphilis occur among children in Russia, where the peasants live huddled together and in ignorance. Syphilis may be passed from one person to another by kissing, and the danger is greater when there are mucous patches or other open lesions upon the mouth. The disease may also be transmitted in wounds inflicted by the teeth of syphilitics. In surgery and midwifery practice physicians are not infrequently infected through minute abrasions—a pin prick or a scratch from a scalpel is sufficient to introduce the virus. Midwifery chancres are usually upon

the fingers. Chancre of the lip is the most common of the erratic or extragenital forms, and may be acquired in many ways apart from direct infection, such as the use of spoons, glasses, pipes, etc., which have recently been mouthed by a syphilitic. The virus may also be transmitted by towels, clothing, razors, handkerchiefs, surgical and dental instruments, human vaccine virus, etc. The list of articles that have conveyed the contagium is comprehensive. The *Treponema pallidum* is a fragile organism and soon dies upon fomites, but the infection is sufficiently prevalent and the danger sufficiently real to demand care. Chancres of the mouth and on the tonsils result, as a rule, from perverted practices. Wet nurses are sometimes infected on the nipple, and it occasionally happens that the relatives of a syphilitic child are accidentally infected. The hereditary and congenital transmission of syphilis is discussed on page 446.

Syphilis lowers the standard of health and paves the way for other diseases. Whatever the etiological relationship may be, it is definitely known that syphilitics are prone to die early from affections of the heart and vessels, general paresis, diseases of the central nervous system (locomotor ataxia), chronic nephritis, arteriosclerosis, aneurysm, etc. The actuaries of all life insurance companies know that the morbidity and mortality rates among syphilitics are very much higher than that of any other class of individuals of the community who enjoy apparent good health at the time of examination.

Most insurance companies refuse to accept syphilitics at all. Some companies require extra premiums to compensate for the extra risks; a few companies will accept exceptionally favorable cases who have had a thorough course of treatment, and who have shown no symptoms for 3 to 5 years, but under these circumstances only special policies are contracted for which do not keep the applicant on the companies' books after 55 years of age.

Syphilis was regarded as an infection peculiar to man until Nicolle and Hamonic in 1902, and Metchnikoff and Roux in 1903, transmitted the disease to the higher apes. As a result of these experiments certain important facts in reference to prophylaxis were discovered. Metchnikoff and Roux found that bichlorid of mercury, 1-2,000, applied one hour after inoculation, does not prevent the development of the primary lesion in the monkey. This is probably due to the fact that the action of the bichlorid is limited to the surface; it lacks penetration owing to its well-known property of coagulating albumin. Other antiseptics were tested, but in a long series of experiments, carried out on chimpanzees, baboons, and Macacus monkeys, Metchnikoff and Roux showed that mercurial inunctions are most successful in preventing the development of the chancre. The mercurial inunctions may be made with metallic mercury, calomel, white precipitate (ammoniated mer-

cury), or salicyl-arsenite of mercury. Calomel ointment appears to be the best, and is the one now generally used. It is rubbed up in lanolin in the proportions of 1 to 3 or 1 to 4. The ointment should be rubbed upon the place for 4 to 5 minutes and not later than 20 hours after the receipt of the infection. This will usually prevent the development of the disease. Excision, or destruction of the chancre with the actual cautery or with corrosive antiseptics does not influence the development of the disease.

GONORRHEA

Gonorrhea is much more prevalent than syphilis, and common opinion regards it as a comparatively trivial infection, that is, "no worse than an ordinary cold." As a matter of fact, gonorrhea is one of the serious infectious diseases, and the gonococcus occupies a position of high rank among the virulent pathogenic microorganisms. From an economic and public health standpoint, gonorrhea does not fall very far short of syphilis in importance; in fact, some give it first place.

The serious consequences of gonorrhea are: complications such as periurethral abscess, gonorrheal prostatitis in the male, and vaginitis, endocervicitis, and inflammation of the glands of Bartholini in the female. Perhaps the most serious of all the sequelæ of gonorrhea are those which result from the spread by direct continuity of tissues, such as inflammation of the Fallopian tube, and sometimes of the endometrium, the ovary, or even the peritoneum. The gonococcus has been found in pure culture in cases of acute general peritonitis. Other inflammations caused by the spread of the infection are cystitis, which sometimes extends upward through the ureters to the kidneys.

The gonococcus sometimes invades the blood and produces a general septicemia and pyemia; death may occur from acute endocarditis. Gonorrheal arthritis is, in many respects, the most damaging, disabling, and serious of all the complications of gonorrhea. It may even follow ophthalmia neonatorum. It is more frequent in males than in females, but a gonorrheal arthritis of great intensity may occur in a newly married woman infected by an old gleet in her husband (Osler). The serious nature of gonorrheal complications in the eye will be considered separately under Ophthalmia Neonatorum. Gynecologists tell us that the greater part of their practice is made up of the consequences of gonorrhea.

Sterility is one of the serious consequences of gonorrhea. This may be caused in the male through epididymitis, which is a very common complication, and in the female by salpingitis, which closes or obstructs the Fallopian tube. Stricture of the urethra in the male is a frequent sequel.

Gonorrhea is usually transmitted by sexual congress; however, accidental or innocent infections are not infrequent. Paul Bendig [1] reports the following instance: Of 40 girls sent for convalescence to a brine bath, 15 showed signs of gonorrhea after the return. The infection came from an eight-year-old girl, who apparently had been suffering from gonorrhea for several years, and was spread through indiscriminate bathing in one bath tub and the use of the same bath towel.

Gonorrheal infections in children require special consideration. The frequency of such infections may be judged from the observations of Pollack, who reports 187 cases treated in the Woman's Venereal Department of Johns Hopkins Hospital during the year 1909.[2] Pollack estimates that 800 to 1,000 children are infected each year in Baltimore, and that the same proportion probably holds good for other cities. The cause of the frequent infection among children is in part the superstition that a person infected with syphilis or gonorrhea may get rid of it by infecting another—especially a virgin.

When gonorrhea enters a children's hospital or an infants' home it is prone to become epidemic and is very difficult to eradicate. The story of the infection in the Babies' Hospital, New York, for eleven years, as told by Holt,[3] illustrates the singular obstinacy of the infection. In spite of the greatest care and precaution, there were, in 1903, 65 cases of vaginitis with 2 of ophthalmia and 12 of arthritis. In 1904 there were 52 cases of vaginitis, only 16 of which would have been recognized without the bacteriological examination. In all, in the eleven years, there were 273 cases of vaginitis; 6 with ophthalmia and 26 with arthritis. Holt urges isolation and prolonged quarantine as the only measures to combat successfully the disease (Osler). It is impossible to control such epidemics without bacteriological diagnosis.

Chancroid is not discussed separately because its prevention is similar to the measures used against syphilis and gonorrhea. Chancroid sometimes directly results in severe, even fatal, results, but does not, as a rule, leave dangerous sequelæ.

VENEREAL PROPHYLAXIS AND HYGIENE OF SEX

The same principles apply to the prevention of the venereal diseases as apply to the prevention of other communicable diseases. The fight against venereal diseases, however, is especially complicated and difficult because of the close association with prostitution, the problems of sex hygiene, and alcoholism—in fact, the question pervades the woof and

[1] *Münchener med. Wochenschr.*, 1909, p. 1846.
[2] *Johns Hopkins Hospital Bulletin*, May, 1909, p. 142.
[3] *New York Med. Jour.*, March, 1905.

warp of society. There are three primitive appetites of man—hunger, thirst, and the sexual appetite. The first two persist throughout life; the last comes on at puberty, grows stronger during adolescence, and wanes with age. Any program for the control of the venereal diseases or the hygiene of sex must take into account the fact that we are dealing with a primal, impulsive, and natural passion which is the greatest force for social good, when used in accordance with the laws of nature, but may result in dire consequences when these laws are transgressed. The venereal diseases are among the most widespread and universal of all human ills, and enter more largely in the making and marring of domestic happiness than any other disease known to man. The difficulties of the situation should not deter the health officer and all those who labor for social uplift, for there is no more pressing problem in preventive medicine.

Attitude.—Our attitude toward the venereal diseases is very inconsistent. There is a natural aversion toward these afflictions. The sanitarian should make no distinction between the venereal diseases and other epidemic diseases; he should regard the greatpox in the same light that he regards the smallpox. The principles for the control of syphilis and gonorrhea differ in no wise from those used to control smallpox, leprosy, tuberculosis, measles, diphtheria, etc. The health officer must not regard venereal disease as a punishment for sin and crime—the victim or culprit needs help and sympathy. The immediate problem is the prevention of further spread of the infection. A person afflicted with a venereal disease should be treated in the same humane spirit that actuates the physician in other diseases. Furthermore, the interests of the community require that the patient be accorded the best possible care and attention. The usual attitude toward the venereal diseases may well startle us when we consider that in most of our large cities no hospital will take a case of syphilis or gonorrhea during the acute stages, when these diseases are especially communicable. Morrow holds that the notoriously inadequate provision made for the reception and treatment of venereal patients is a disgrace to our civilization. Formerly lepers were segregated in vile lazarettos and cases of smallpox isolated in horrible pest houses; now we have comfortable and congenial isolation wards or special sanatoria for these diseases. From the standpoint of prevention suitable hospital accommodations should be provided for the venereal diseases.

Education.—Education in sex hygiene and the venereal peril accomplishes a certain amount of good. It may be questioned how much a knowledge of the consequences will prevent some persons committing crime. However, the old-style innocence must be regarded as present-day ignorance. Every boy and girl, before reaching the age of puberty, should have a knowledge of sex, and every man and woman be-

fore the marriageable age should be informed on the subject of reproduction and the dangers of venereal diseases. Superficial information is not true education. On the other hand, it is a mistake to dwell unduly upon the subject, for in many instances the imagination and passion of youth are inflamed by simply calling attention to the subject. One of the objects of education is to avoid the dangers of sex impurities, and all agree that this may often best be accomplished by keeping the mind clean, that is, away from the subject. The education must, therefore, be clear, pointed, brief, and direct. The object of education is not alone to help the individual to help himself, but to influence necessary legislation and concerted public action: also to lessen the influence of quacks. A simple knowledge of the facts is a sufficient deterrent for some; others may be influenced through fear of the consequences.

In general, it may be said that the best plan of education in matters sexual is to answer the questions of children upon the subject of maternity frankly and truthfully, but to offer them no information on the subject. The growing child at the age of puberty should be offered a certain amount of information concerning unnatural habits and should study physiology, biology, especially botany, and the facts of fertilization. At about the age of sixteen or eighteen girls as well as boys should be instructed as to the venereal peril. The pamphlets issued by the Committee on Sex Hygiene of the Massachusetts Association of Boards of Health are admirable. One circular is for young men, another for young women, and a third for those having venereal disease.

Some of the facts all young men should know are: that the true purpose of the sex function is reproduction and not sensual pleasure; that the testicles have a twofold function, (a) reproduction and (b) to supply force and energy to other organs of the body; that occasional seminal emissions at night are evidences of normal physiological activity; that sexual intercourse is not essential to the preservation of virility; that chastity is compatible with health; and that the sex instinct in man may be controlled.

The primary function of the testicles is to build the boy into the man. Castration in early life, as in the case of eunuchs, results in a loss of the internal secretion of the testicles and a failure in development of the secondary sexual characters which distinguish the male. There are an alteration in physical conformation and in the voice, lack of beard, development of the mamma, etc.—in other words, an approach to the feminine type. Healthy sexuality stimulates the imagination, sentiment, the esthetic sense, and the higher creative functions. Excesses or any influence which weakens the sexual system impair the will power, influence self-respect, and diminish mental force. Experience shows that arduous physical and mental labor, even after maturity

is attained, is best performed when the sex organs are not exercised; while sexual excess distinctly impairs muscular strength and mental efficiency. It is unwise to frighten boys by exaggerating the results of self-abuse, which is rather the effect and not the cause of idiocy, insanity, degeneracy, and other defects of the central nervous organization. Self-abuse is no worse in its effects than natural coitus, except for its influence upon character. Both are alike harmful when indulged in to excess.

Registration of Cases.—It is not possible to control any communicable disease, especially one that is pandemic, such as syphilis or gonorrhea, without a knowledge of the cases and deaths. It is perhaps even more important to collect morbidity and mortality statistics of the great-pox than it is of the smallpox. But the public registration of private disease at once defeats its own object. Compulsory methods have heretofore failed, and little may be expected from voluntary registration. When we consider that in our country we have no means of knowing the amount and distribution of smallpox, except to a limited degree in the registration area (which is less than one-third of our domain), what can we expect from the registration of the closely guarded secrets of the underworld? The public registration of ophthalmia neonatorum is successful because this form of gonorrhea is so apparent and the consequences so immediate and serious. The difficulties, however, need not deter us, and registration should be attempted even though the returns are incomplete. A start should be made, and, though the returns will be only partial at first, a gradual improvement may be expected. Every case known and properly cared for is a focus of infection neutralized.

Continence.—One of the important facts to teach boys is that continence is compatible with health. The testicles are like the tear glands and the sweat glands, in that they do not atrophy with disuse. Benjamin Franklin taught, as many another man of influence believes today, that the exercise of the sexual functions is necessary for health. This is a mistake and has done much harm.

The sex principle is universal in nature. It is the force behind the constructive and progressive processes of all life, from the color adaptations of birds and flowers to the highest leadership in men. Reproduction is only one of its many functions; and the man who assumes that the so-called physical desire that at times thrills him indicates a need of sexual intercourse is in danger of depleting and wasting from his life a chief source of physical and mental growth.

The single standard for men and women must be insisted upon, and the parent or guardian is justified in demanding a clean bill of health of the young man who proposes marriage. The young man, in turn, is entitled to the same from his prospective father-in-law. One of the

defects of our artificial civilization which leads to harm is the postponement of the marriage age.

Carnal lust may be cooled and quelled by hard work of the body, as well as attention to personal hygiene—hence, one of the great advantages of athletic sports for growing young men.

Personal Hygiene.—Idleness, stimulating food, overeating, impure thoughts, evil associates, and alcohol excite the passions and are the bedfellows of the venereal diseases. Purity of mind and cleanliness of body are helpful prophylactics. Physical exercise and an out-of-door life divert the mind and help the body; it is a good safety valve for the excess animalism of youth.

The public should be taught the necessity for thorough daily cleansing of the external genitals in both sexes, even in children. The large number of secreting glands and the decomposition of their secretions are liable to induce irritation and even minute lesions which open portals to infection of all kinds.

Alcohol.—The strongest indictment against alcohol is that it excites the passions and at the same time diminishes the will power. The fact that alcohol lowers moral tone does much more harm than all the cirrhotic livers, hardened arteries, shrunken kidneys, inflamed stomachs, and other lesions believed to be caused by its excessive use.

Prostitution.—The regulation of prostitution by means of medical inspection has been tried and largely abandoned. In other words, it is a failure, for the reason that it makes vice easy and is, therefore, morally wrong. It gives a false sense of security and does not reach clandestine prostitution, which is the great source of the venereal diseases. Under certain limited conditions, such as in army encampments, where clandestine prostitution can be eliminated, regulation has markedly diminished the prevalence of venereal disease.

The elimination of prostitution is beyond the dream of even the theoretical reformer. Its control resolves itself into questions of personal hygiene and public hygiene; it is inextricably mixed up with alcoholism, and, like the abuse of alcohol, the question may best be reached by that slower, surer process of improving the moral and physical fiber of man.

Medical Prophylaxis.—In accordance with the researches of Metchnikoff and Roux a reasonably efficient prophylaxis against the venereal diseases is now possible. In the United States Navy the following method is employed: The entire penis is scrubbed with liquid soap and water for several minutes, and then washed well with a solution of mercuric bichlorid, 1 to 2,000 in strength. If there are any abrasions present, they are sprayed with hydrogen peroxid from a hand atomizer. The man is then placed in a sitting position, well forward in a chair in front of a convenient receptacle, and given two injections of a 10

per cent. solution of argyrol. He is required to retain each injection in the urethra for five minutes. After taking the injections, the entire penis is thoroughly anointed with a 33 per cent. calomel ointment. He is told not to urinate for at least two hours, and to allow the ointment to remain on the penis for some hours. A temporary dressing is placed on the parts to protect his clothes.

The measures which will prevent gonorrhea will not ward off syphilis, and *vice versa.*

The results attending such prophylactic treatment are very good. Thus Ledbetter [1] reports that at Cavite, before medical prophylaxis was instituted, the percentage of venereal diseases of all classes among the men averaged from 25 to 30 per cent. annually, and at times even higher. The percentage of gonorrhea was reduced to 8 per cent. annually, and this percentage included about 30 patients who did not report for treatment. Chancroid was reduced from 5 to 2 per cent., which included 2 patients not reporting for treatment. Syphilis has been reduced from about 20 cases annually to one case for the entire year 1910, and this patient did not report for prophylactic treatment. The results speak for themselves and show the efficiency of the prophylactic measures if properly and thoroughly carried out.

Holcomb and Cather [2] report the following as a result of treatment used by them in 3,268 persons in the U. S. Navy between May 1, 1910, and August 31, 1911. The experience is considered to be a fair index of the results of medical prophylaxis. The treatment used by them is as follows: (1) Wash the penis, head, shank, and under frenum with 1-5,000 bichlorid of mercury solution with a cotton sponge. (2) Pass water. Take urethral injection of 2 per cent. protargol solution and hold to count 60. (3) Rub 50 per cent. calomel ointment well into foreskin, head, and shank of penis, with particular care about the frenum. Treatment taken within eight hours after exposure in 1,385 cases shows 19 infections, or but 1.37 per cent. In the interval of from eight to twelve hours after exposure in 741 cases shows 25 infections, or 3.31 per cent. Between twelve and twenty-four hours in 920 cases shows 46 infections, or 5 per cent. Of the 56 cases of gonorrhea occurring in the first twenty-four-hour interval, 26 were recurrent cases; the remaining 30 were primary infections.

The use of salvarsan early in syphilis will prevent the further spread of the infection.

Segregation.—Theoretically, every case of syphilis or gonorrhea should be isolated until the danger of infection is passed. Practically,

[1] Ledbetter, Robert E., "Venereal Prophylaxis in the U. S. Navy," *Jour. A. M. A.*, April 15, 1911, Vol. LVI, No. 15, p. 1098.

[2] Holcomb, R. C., and Cather, D. C., U. S. N., "Study of 3,268 Venereal Prophylactic Treatments," *Jour. A. M. A.*, Vol. LVIII, No. 5, Feb. 3, 1912, p. 368.

however, segregation is impracticable except with a limited number of cases. With better and more attractive hospital facilities and free beds a certain amount of segregation may be accomplished voluntarily and humanely. An alert health officer can trace the source of infection in certain cases and induce the women responsible to take the salvarsan treatment in the case of syphilis, or to submit to hospital care in the case of gonorrhea or chancroid.

Routine circumcision and a medical examination as a necessary preliminary to marriage are further hygienic reforms advocated.

Finally, in considering venereal prophylaxis, it should be remembered that these diseases are of great antiquity and seem likely to continue indefinitely, that they already affect a large number of the population, and are spreading; that the existing means for the treatment of them among the poor is insufficient; that the common mode of propagation is irregular and illicit intercourse; that prostitution arose in response to the strongest instincts and passions in the human breast; and that prostitutes themselves need protection and have claims on the humanity of the law.

PREVENTABLE BLINDNESS

Preventable blindness is considered in this place because the largest single factor causing needless loss of eyesight is gonorrhea. Among the infectious eye troubles the most destructive is ophthalmia neonatorum.

There are 64,000 registered blind persons in the United States. Of these about 10 per cent. (between six and seven thousand) are blind as the result of ophthalmia neonatorum. From 25 to 30 per cent. of all the blind children in all the blind schools of this country owe their infliction to gonorrhea. It has been estimated that probably one-half of the blindness in the world is preventable.

Emphasis upon the great harm done by ophthalmia neonatorum should not blind us to the fact that there are other causes of blindness and eye deterioration which are preventable; thus we have to consider the later pus infections, syphilis, sympathetic inflammations, industrial accidents, accidents at play, progressive nearsightedness caused by violation of ocular hygiene, and a variety of inflammatory conditions. Functional disturbances of vision (amaurosis) and atrophy of the optic nerve may be brought about by poisoning with lead, alcohol, tobacco, and other toxic substances. This form of dimness of vision, or even loss of sight, occurs rather frequently, and in many instances is preventable.

Trachoma is a menace to the integrity of sight. It is an infection

caused by a filterable virus.[1] It flourishes best where sanitary conditions are worst. The disease is slow and insidious in its development. A mass of sago-like granulations gradually fills in the retrotarsal fold, thereby limiting the lid movements and leaving the eye half closed. The infection is rubbed into the eye by roller towels, handkerchiefs, fingers, and other ways. When once established, the disease is chronic, and permanent cures are doubtful. Trachoma is much more prevalent in the United States than ordinarily supposed. The public eye clinics of Chicago are filled with patients showing the resulting deformities. Wilder located a center in southern Illinois, and it has also been found in the mountains of Kentucky and Tennessee, while in Oklahoma it has become a public menace. It is more or less prevalent in the poorer sections of all the large centers.

Trachoma is of such a serious nature that all immigrants arriving at our shores have their eyelids everted and conjunctivæ examined for evidence of this infection. An alien with trachoma is deported and the steamship is liable to a fine of one hundred dollars for bringing every case of trachoma where it can be shown that the disease might have been recognized at the port of departure.

Wood alcohol is one of the causes of blindness. As small a quantity as a teaspoonful has caused loss of vision. Wood alcohol is used as an adulterant, especially in liquors. The excessive use of tobacco also leads to dimness of vision.

In New York State about 200 industrial accidents resulting in total blindness occur annually. Besides this, there is a large number of accidents occurring on railroads in construction work, and in the field and forest. Many of the accidents to the eyes occurring in factories are preventable. The majority of such accidents are due to small flying particles.

A material proportion of blindness is caused by accidents to children at play; sometimes the eyeball is torn by a buttonhook or pierced by a knife or awl; or a scissors blade, used to untie a knot, slips. Some eyes have been injured by the crack of a whip, by a shot from an air-gun or toy pistol. Accidents also occur to the eyes from fireworks, especially on the Fourth of July.

OPHTHALMIA NEONATORUM

Ophthalmia neonatorum or inflammation of the eyes of the newborn includes all the inflammatory conditions of the conjunctiva that occur shortly after birth—usually before the end of the first month.

[1] Bertarelli and Cacchetto, *Centr. für Bakt.*, Orig., I Abt., Bd. XLVIII, 1908, p. 432.

The conjunctivæ of the newborn are peculiarly liable to infections. This delicate membrane rapidly acquires an immunity of a high order. The gonococcus is usually the cause of severe conjunctivitis occurring in a baby a few days old. The gonococcus has been demonstrated in 65 per cent. of all cases, mild and severe.

Ophthalmia neonatorum is not always gonorrheal, but may be produced by other virulent microorganisms or by irritating substances. The microorganisms other than the gonococcus that sometimes cause conjunctivitis during the early days of life are: streptococci, the meningococcus, the Koch-Week's bacillus, the pneumococcus, the diphtheria bacillus, and even staphylococci. These are relatively so rare that we may disregard their etiological significance for our present purpose. The diagnosis of gonorrheal ophthalmia may readily be made by simply examining a stained smear of the secretion.

The infection commonly occurs during the passage of the child through the genital tract of the mother and usually just before delivery. It is caused by the entrance of the vaginal secretion containing gonococci into the conjunctival sac. It may also be caused after delivery by infected hands, towels, sponges, or other objects.

The disease varies in severity; sometimes it is very mild, with slow onset and spontaneous recovery. Usually, however, it is severe and serious. The inflammation may extend from the conjunctiva to the cornea and invade the deeper structures of the eye. Corneal ulcers and opacity may result, with complete loss of vision. In a typical case both the ocular and palpebral conjunctivæ are red and very much swollen; the eyelids and surrounding tissues are infiltrated and there is a thick, creamy, abundant secretion.

There are many grades of mild inflammatory condition, which must not be mistaken for gonorrhea. At birth the eyelids are almost always glued together with the normal sticky secretions. It is common, too, for the lids to remain red and sticky for a day or so. The diagnosis may be made in a few minutes by a microscopic examination.

Prevalence.—Kerr calls attention to the fact that there are no complete statistics showing the prevalence of ophthalmia neonatorum, and only an approximate idea can be had of the number of cases by studying the admissions to schools for the blind. A committee of the British Medical Association found that more than one-third of those in blind schools of Great Britain owed their affliction to this disease.[1]

In the United States and Canada, in 1907, out of 224 admissions to 10 schools for the blind, 59, or 24.38 per cent., were blind as a result of ophthalmia neonatorum;[2] and out of 351 admissions to certain

[1] *British Medical Journal*, May 8, 1909.
[2] *Jour. A. M. A.*, May 23, 1909, p. 1745.

schools in the United States and Canada in 1910, 84, or 23.9 per cent., were blind from this cause.[1]

As a result of studies made of ophthalmia neonatorum in 10 manufacturing cities of Massachusetts, Greene has presented figures which show that the minimum morbidity rate for this disease was 6.4 per 1,000 births. A more complete census made by him for the practice of 173 physicians in 9 cities revealed an average morbidity rate of 10.8 per 1,000 births.[2]

It is estimated that the total annual loss from gonorrheal ophthalmia in the United States is seven million dollars, and that an amount of more than one million dollars annually is spent in partially caring for its victims. A blind child costs the community an excess of about $4,500 for its schooling.

Prevention.—Credé's Method.—Credé in 1881 introduced an efficient method of preventing ophthalmia neonatorum at the Lying-in Hospital at Leipzig, thereby connecting forever his name with the prevention of the disease and the subsequent saving of the sight of many infants. Credé's original method consisted simply in placing one or two drops of a 2 per cent. solution of silver nitrate in each conjunctival sac, as soon as practicable after the birth of the head.

In order to prevent gonococcic as well as other infections of babies' eyes, the following procedure is recommended: During pregnancy women should be instructed to practice daily external cleansing with soap and water and a clean wash-cloth. In case of any irritating discharge or even profuse white discharge, a physician should at once be consulted.

Immediately after labor the eyelids should be carefully cleaned with sterile absorbent cotton or gauze and a saturated solution of boracic acid. A separate pledget should be used for each eye and the lids washed from the nose outward until quite free of all mucus, blood, or meconium without opening the lids. Next the lids should be separated and one or two drops of a one per cent. silver nitrate solution should be dropped into each eye, between the outer ends of the lids. The lids should be separated and elevated away from the eyeball so that a lake of silver nitrate solution may lie for one-half minute or longer between them, coming in contact with every portion of the conjunctival sac. One application only of the silver nitrate should be made, and ordinarily no further attention need be given to the eyes for several hours. Each time the child is bathed the eyes should first be wiped and cleaned with pledgets of sterile absorbent cotton wet with a saturated solution of boracic acid.

[1] *Ibid.*, July 1, 1911, p. 72.
[2] *Monograph Series of the American Association for Conservation of Vision*, Vol. I, No. 1.

Credé used a 2 per cent. solution of silver nitrate, but, as this is sometimes irritating, a 1 per cent. solution is now commonly employed, and seems to afford equally efficient prophylaxis. The silver nitrate solution should be instilled into each conjunctival sac but once. Repeated applications may cause serious inflammations. In fact, a single treatment sometimes causes a conjunctivitis, known as "silver catarrh." Because of the silver catarrh the strength of the silver nitrate solution has not only been reduced from a 2 to a 1 per cent. solution, but this may be neutralized after instillation with salt solution. Other prophylactic substances have been proposed. The best substitutes are a few drops of the newer silver compounds, as argyrol (25 per cent.) or protargol (5 per cent.). The following have also been recommended: Bichlorid of mercury, 1-2,000 or 1-5,000, silver acetate, 0.23 per cent., recommended by Zweifel, who used it in 5,222 cases. Schmidt and Rimpler recommend *aqua chlorini*. Carbolic acid (1 per cent.) or other antiseptics have also been tried. No substance, however, is known to be as reliable as silver nitrate, which should be used in all cases where there is any reason for believing that the mother is infected with the gonococcus.

If a conjunctivitis is present, a bacteriological examination of the discharge should at once be made. If the inflammation is due to the gonococcus a 2 per cent. silver nitrate solution should be used. In certain mild, non-gonorrheal infection 0.5 per cent. is usually sufficient. If the Klebs-Loeffler bacillus is found, diphtheria antitoxin should be given without delay. If the diplococcus is present, a weak solution (1 grain to the ounce) of zinc sulphate should be instilled frequently.

As a general rule, it is advisable to use a prophylactic as a matter of routine in hospital and private practice. To use Credé's method upon every case necessitates the unpleasant suspicion that every woman is a possible source of gonococcus infection. If statements of the father about his previous life can be relied upon, an eye prophylactic can be safely omitted. In his private work Williams uses a boric acid solution except where there is special reason for believing that the mother has gonorrhea. The responsibility for risking the baby's eyes rests upon the medical attendant. There can only be one safe rule in case of doubt. It should be remembered that gonococcic infections of the conjunctiva occur in about one to every two hundred births (Edgar).

The good results of Credé's method are sufficiently convincing to justify criminal proceedings upon those who fail to apply this simple prophylactic. Haab reduced the frequency of ophthalmia neonatorum in hospital practice from 9 to 1 per cent., while the statistics of many hospitals show only a very small fraction of 1 per cent. Stephenson's results are typical. In 2,265 births, ophthalmia neonatorum developed in 10 per cent. of the cases preceding the use of Credé's method. In

1,160 births after this method only 0.17 per cent. developed any trouble. A small number of cases may develop despite the use of silver nitrate.

The technique of applying the nitrate of silver is very important, for, in the opinion of Edgar, when ophthalmia neonatorum develops after the use of nitrate of silver, it is due either to a secondary infection or to the fact that the solution does not really bathe the mucous membranes, but remains upon the lashes. The lids must be everted and the silver solution placed in the conjunctival sac either from a glass rod or a pipette. Care must be taken not to touch or injure the delicate membrane.

Credé's method does not strike at the root of the evil. It would, of course, be much better to eradicate gonorrhea from men and women than to be compelled to drop silver nitrate into babies' eyes. Wrapped up with the question of ophthalmia neonatorum is the question of midwives, for to prevent blindness we must have intelligent and conscientious obstetrical attendants, especially for the poor and ignorant classes. Midwifery practice needs regulation, supervision, and elevation. Education is one of the bulwarks of prevention in this as well as other preventable infections.

Legislation.—Ophthalmia neonatorum is an instance in which "the protection of the citizen from the assaults of ignorance, indifference, or neglect, when they threaten his well-being and even his economic efficiency, is a duty which the state cannot evade and which he has a right to exact."

Laws for the prevention of the blindness of newborn infants are making progress slowly. Among the states in which the disease is notifiable are Connecticut, Massachusetts, Minnesota, Nebraska, New York, Oregon, South Carolina, Utah, Vermont, and Wisconsin. In some states the nurse, midwife, or parent is required to report the disease, in other states the attending physician.

Maine was the first state to take legal steps in 1891 to control ophthalmia neonatorum. In 1892 New York followed, with an amendment to the law relative to midwives and nurses. Subsequently most of the other states took legislative action.[1] The provisions of the several laws are quite varied. In all of them, however, the object is to insure early treatment, and to this end compulsory notification is generally required. The health authorities of Massachusetts, New Jersey, Vermont, Rhode Island, New York, and the District of Columbia furnish prophylactic outfits to physicians. The outfit ordinarily consists of a small vial containing a 1 per cent. solution of nitrate

[1] Kerr, J. W., "Ophthalmia Neonatorum: An Analysis of the Laws and Regulations Relating Thereto in Force in the United States," *Public Health Bull. No. 49,* U. S. P. H. & M. H. S., Oct., 1911.

of silver, a sterilized dropper and bulb, and a circular of instructions.

In order to make material progress against ophthalmia neonatorum, as well as against infant mortality, it is essential that laws require prompt report of all births; it is the duty of the health authorities to see to it that such laws are effectively carried out.[1]

TETANUS

Compared with the major plagues of man, lockjaw has always been a rare disease. It is on account of the characteristic and fatal spasms that it early attracted attention. The student will be well repaid by a study of the historical development of the theories that have been advanced since the time of Hippocrates to explain the cause of tetanus. These theories mirror the prevailing thought upon the nature of disease as it developed from that of evil spirits, through the humoral theory, the realm of miasms and noxious effluvia, to the germ theory. Tetanus could not escape the rheumatism theory which has been such an alluring catchall for symptoms and diseases difficult of explanation. "Taking cold" was assigned its usual rôle here as elsewhere. When no assignable cause seemed at hand, the disease was given the learned title—idiopathic tetanus.

Etiology.—In 1889, with the aid of anaerobic technique, Kitasato[2] for the first time grew the tetanus bacillus in pure culture, and by

[1] The Massachusetts law reads as follows:

Section 49. . . . Should one or both eyes of an infant become inflamed, swollen and red, and show an unnatural discharge at any time within two weeks after its birth, it shall be the duty of the nurse, relative, or other attendant having charge of such an infant to report in writing within six hours thereafter, to the board of health of a city or town in which the parents of the infant reside, the fact that such inflammation, swelling, and redness of the eyes and unnatural discharge exist. On receipt of such report, or of notice of the same symptoms given by a physician as provided by the following section, the board of health shall take such immediate action as it may deem necessary in order that blindness may be prevented. WHOEVER VIOLATES THE PROVISIONS OF THIS SECTION SHALL BE PUNISHED BY A FINE OF NOT MORE THAN ONE HUNDRED DOLLARS.

Section 50. . . . If a physician knows that . . . if one or both eyes of an infant whom or whose mother he is called to visit become inflamed, swollen, and red, and show an unnatural discharge within two weeks after birth of such infant, he shall immediately give notice thereof in writing over his own signature to the selectmen or board of health of the town; AND IF HE REFUSES OR NEGLECTS TO GIVE SUCH NOTICE, HE SHALL FORFEIT NOT LESS THAN FIFTY NOR MORE THAN TWO HUNDRED DOLLARS FOR EACH OFFENCE. (Revised Laws, Chapter 75.)

[2] *Zeitschr. f. Hyg.*, Vol. VII, 1889, p. 225.

successful inoculation experiments proved that this bacillus was the real cause of tetanus. Kitasato further showed that the tetanus bacillus is not found in the heart's blood of mice dead of tetanus, and therefore concluded that we are dealing with an intoxication, and not an infection. We now regard tetanus as a type of the true toxemias. This work of Kitasato's was one of great importance, and led up to the epoch-making discovery of Behring and Kitasato [1] in the following year (1890) upon tetanus and diphtheria toxines and antitoxins, laying the foundation of serum therapy.

Tetanus may be regarded almost solely as a wound complication. All wounds are not equally liable to this complication, even though tetanus spores are present. Punctured, lacerated, and contused wounds are much more susceptible to tetanus than cleancut or superficial wounds. The size of the wound is of much less consequence than its character. Fatal tetanus may develop from trivial wounds, such as pin scratches, small splinters, insect bites, vaccinations, etc.

Symbiosis is an important factor in tetanus. Wounds infected with pyogenic organisms and other bacteria favor anaerobic conditions and permit the tetanus spores to germinate, and seem to encourage the growth of the bacillus and the development of toxine.[2] A few tetanus spores free of tetanus toxin in a clean wound may be taken care of by the phagocytic cells. This may readily be demonstrated experimentally by injecting animals with tetanus spores washed free of toxine.

The normal habitat of tetanus is in the intestinal tract of herbivorous animals. Sanchez, Toledo, and Veillon [3] found tetanus in the feces of 4 out of 6 horses and in the feces of 1 of 2 cows. Park found tetanus bacilli in the intestines of about 15 per cent. of horses and calves living in the vicinity of New York City. They are present to a somewhat less extent in the intestines of other animals and of man.

It is rather a curious paradox that the horse, which is the most susceptible of all animals to tetanus toxin, is one of the principal hosts of the tetanus bacillus.

The spores taken in the food are not affected by gastric digestion, and in the small intestines find ideal anaerobic conditions, food supply and temperature for growth and development. Here they very probably multiply and pass in the dejecta to pollute the soil. The soil, therefore, in all regions inhabited by man and domestic animals is more or less contaminated with tetanus. The bacilli, however, do not multiply in the soil. While the soil acts only as a vehicle, it is the immediate source of the large proportion of tetanus spores.

[1] *Deutsch. med. Wochens.*, Vol. XVI, No. 40, p. 1113.

[2] In the laboratory some of the strongest tetanus toxins have been prepared from mixed or contaminated cultures.

[3] *La Semaine Méd.*, 1890, X, p. 45.

It is assumed, but not proven, that tetanus bacilli grow in the intestinal tract of herbivora. It is conceivable that the spores simply pass through the intestines without multiplying at all, but it is known that tetanus is capable of multiplying in symbiotic relation with other bacteria wherever protein matter undergoes putrefaction under anaerobic conditions.

On account of the great resistance of the spores, they are blown about in dust and are spread everywhere by dirt and manure. Tetanus has been found in hay dust, on horses' hair, in the dust of houses, barracks, and hospitals, in the mortar of old masonry, in street dust, in gelatin, and in the greatest variety of places.

One of the agencies in the distribution of tetanus spores over limited areas is undoubtedly the common house fly. The arrow heads of certain savages in the New Hebrides contain tetanus spores obtained by smearing the arrowheads with dirt from crab holes in the swamps (Le Dantic).

Tetanus bacilli are not equally numerous in all localities. The infection is much more prevalent in warm than in cold countries. It is especially severe in the tropics, yet Iceland at one time suffered severely from tetanus neonatorum. Some parts of Long Island and New Jersey have become noticeable for the number of cases of tetanus caused by small wounds. Tetanus spores are widely disseminated in India. Goodrich states that in Bombay alone there were 1,955 cases of tetanus in five years. These do not include the puerperal cases.

Tetanus occurs either sporadically or in epidemic form. Formerly epidemics in hospitals (especially in lying-in hospitals) and in wars were rather common. Before the days of antisepsis the infection was readily spread through instruments, fingers, bandages, etc.

Trismus neonatorum, or tetanus of the newborn, was a common and very fatal infection, especially in the tropics. Before the days of asepsis the infection was permitted to enter through the umbilical wound. In certain of the West Indian islands more than one-half of the mortality among the negro children has been due to this cause. Since the introduction of proper methods of treating the cord the disease is rare.

The wounds produced by blank cartridges are especially liable to develop tetanus. The source of the tetanus spore in these cases is not entirely clear. Wells examined 200 cartridges from five firms without finding the tetanus bacillus. It is probable that the spore is upon the skin and is carried along with the paper and powder from the blank cartridge. The peculiar character of the wound favors the development of tetanus.

The great decrease in the number of cases of tetanus following Fourth of July wounds is due to the vigorous campaign carried on

by the American Medical Association. In 1903 there were 406 deaths from tetanus; in 1904, 91; 1905, 87; 1906, 75; 1907, 73; 1908, 76; and in 1911 only 18 cases and 10 deaths. Eighty per cent. of these followed blank cartridge wounds. The good results are attributed to the more thorough and careful treatment of the wounds and especially the use of tetanus antitoxin as a prophylactic—and more recently to safer and saner methods of celebration.

Tetanus spores or toxine may contaminate bacterial vaccines, antitoxic sera, vaccine virus, and other biologic products used in human therapy. The possible association of tetanus with bacterial vaccines was demonstrated in the unfortunate outbreak at Mulkowal, India, in 1902.[1] One hundred and seven persons were inoculated with Haffkine's plague prophylactic. Of these 19 were affected with symptoms of tetanus and died. In this case the tetanus probably grew as a contamination in the plague culture, for it is now well known that the anaerobic conditions produced in *B. diphtheriæ*, *B. pestis*, *B. subtilis*, and other organisms in liquid culture media favor the growth of tetanus and the development of its toxin.

In St. Louis (1901) diphtheria antitoxin was taken from a horse during the period of incubation of tetanus and used in amounts from 5 to 10 c. c. upon 7 children, all of whom died of tetanus. Bolton, Fisch, and Walden[2] found that the serum was sterile, but contained tetanus toxin in considerable amount. If the serum had first been tested upon animals, its poisonous properties would have been discovered. This test is now required by the United States law of July 1, 1902, for all serums and vaccines sold in interstate traffic. As a further precaution against this complication horses undergoing treatment for the production of immune sera are given prophylactic doses of tetanus antitoxin from time to time.

Tetanus sometimes occurs as a complication of vaccination. It is not clear in these cases whether the tetanus spores are contained in the vaccine virus or subsequently enter the wound. In many hundreds of special examinations made in the Hygienic Laboratory at Washington tetanus spores have not been found in a single vaccine virus. Experiments show that in vaccine virus purposely contaminated the tetanus spores remain alive and active for a long time (see page 19).

It is, of course, not the rust on a nail that is dangerous, so far as tetanus is concerned, but the spore-bearing dirt it carries into the deep, contused wound that causes the trouble. Gelatin may contain tetanus spores, and the subcutaneous injection of imperfectly sterilized gelatin as a hemostatic has sometimes resulted in accidents.

[1] *Jour. Trop. Med. and Hyg.*, 1907, X, p. 33.

[2] Bolton, Fisch, and Walden in *St. Louis Medical Review*, Vol. XLIV, No. 21, Nov. 23, 1910, p. 361.

Tetanus is harmless when taken by the mouth. Susceptible animals may be given enormous doses of tetanus toxine by the mouth without producing the disease. The bacillus and its spore may be regarded as a saprophyte in the intestinal tract. There is, however, a suspicion that tetanus spores sometimes invade the organism through small wounds in the digestive or respiratory tract. Perhaps some of the cases following surgical operations may be accounted for in this way rather than by infection of the catgut used for ligatures.

Tetanus sometimes occurs in which no wound can be found. This is the so-called "idiopathic or rheumatic tetanus." One explanation of these cases is to be found in the fact that the spores are numerous in street dust and may enter the respiratory tract. They cannot do harm so long as the mucous membrane is healthy, but may enter through inflamed membranes or through small wounds in the nose. Tetanus bacilli have been found in the bronchial mucus of idiopathic cases. Tetanus spores have recently been found in the lymph glands, liver, and other parts of the body, upsetting our previous view that they are strictly confined to the site of the wound. These spores may remain latent for a long time, awaiting favorable conditions to grow and produce toxin, thus giving another plausible explanation of some cases of idiopathic tetanus.

Incubation.—The period of incubation in man is usually from 6 to 14 days. The period is directly proportional to the amount of toxin and the severity of the disease. This can readily be demonstrated upon susceptible animals. In a study of 600 serial tests, Rosenau and Anderson found this direct relation between the period of incubation and the severity of symptoms by the subcutaneous injection of varying amounts of toxin into guinea-pigs. Thus, guinea-pigs that showed symptoms on the third day usually died, a very small percentage recovering. The smaller the dose the longer the onset of symptoms is delayed, the milder is the disease, and the greater the chances of recovery. With a short period of incubation, 6 days or less, the disease in man is almost invariably fatal. With longer periods the disease is milder and recovery frequently takes place without the use of antitoxin or other measures. Tetanus toxin travels up the axis cylinders of the nerves to the cord and brain. It is also distributed in the blood. The period of incubation, therefore, depends somewhat upon the point of entrance of the poison and its proximity to large motor nerve endings.

Resistance.—The tetanus bacillus is readily destroyed by all the ordinary agencies that kill spore-free bacteria. It is killed almost at once in contact with the free oxygen of the air. On the other hand, few, if any, forms of life have a greater resistance than the tetanus spore. Hours of exposure to 60° or 70° C. do not affect them. They usually survive an exposure of one hour to 80° C., but, as a rule, are killed in

streaming steam or boiling water in 60 minutes. Tetanus spores, however, vary greatly in the power to resist the boiling temperature. Kitasato[1] found them to resist 80° C. for one hour, but to be killed in streaming steam in 5 minutes. Vaillard and Vincent[2] found that the spores heated in the presence of moisture in a closed vessel would resist destruction at 80° C. for 6 hours, at 90° C. for 2 hours, and 100° C. 3 to 4 minutes, that they were not always destroyed in 5 minutes, but never resisted more than 8 minutes at 100° C. Levy and Bruns[3] found that destruction begins at 8½ minutes at 100° C.; after 15 minutes few survive, after 30 minutes none. Falcioni[4] studied the subject in view of the dangers of the subcutaneous injection of gelatin. He impregnated gelatin with spores of tetanus bacilli grown in agar or broth for 10 or 12 days, and used Koch's steam sterilization. He found the spores to resist destruction for 2½, but not for 3, hours in streaming steam.

The experimental results are, therefore, sufficiently varied and conflicting to suggest that races of tetanus bacilli exist, the spores of which vary widely in their resistance to moist heat at 100° C. Theobald Smith[5] found that under certain conditions of cultivation some tetanus spores survive a single boiling or streaming steam for 20 minutes regularly, usually for 40 minutes, and occasionally for 60 minutes; in one case 70 minutes' exposure did not destroy the spores. He also showed the possibility of tetanus spores surviving in culture fluids sterilized by discontinuous boiling or steaming in routine laboratory work for fully 20 minutes on three successive days.

Tetanus spores resist the action of 5 per cent. carbolic acid for 10 hours, but are killed in 15 hours. A 5 per cent. solution of carbolic acid, however, to which 0.5 per cent. of hydrochloric acid has been added, destroys them in 2 hours. Bichlorid of mercury, 1-1,000, kills the spores in 3 hours, and in 30 minutes when 0.5 per cent. of hydrochloric acid is added to the solution. According to Park, silver nitrate solution destroys the spores of average resistance in 1 minute in 1 per cent. solution, and in about 5 minutes in a 1 to 1,000 solution. Tetanus spores are destroyed with certainty when exposed to dry heat at or above 160° C. for one hour, or to steam at 120° C. for 20 minutes. Entire confidence may be placed upon either of these two methods.

Direct sunlight does not kill the spores, but seems to diminish their virulence. Under certain circumstances they may live a very long time; Henrijean reports that, by means of a splinter of wood which once

[1] *Zeitschr. f. Hyg.*, VII, p. 225.
[2] *Annales de l'Institut Pasteur*, 1891, V, p. 1.
[3] *Grenzgeb. d. Med. u. Chir.*, 1902, X, p. 235.
[4] *Annali d'igiene sperimentale*, 1904, N. S., XIV, p. 319.
[5] *Jour. A. M. A.*, March 21, 1908, Vol. L, pp. 929-934.

caused tetanus, he was able after 11 years again to cause the disease by inoculating an animal with the infective material.

Prophylaxis.—*Local Treatment of Wounds.*—Wounds, however insignificant, should be thoroughly cleansed. Punctured or lacerated wounds in which there is special danger of tetanus should be freely opened, and every particle of foreign matter carefully removed. Promptness in cleansing the wound surgically is almost as important as thoroughness. Wounds containing garden earth, street dust, or other material liable to contain tetanus spores should receive special consideration. After laying open and thoroughly cleansing such wounds, it may be advisable to disinfect them with the actual cautery or strong chemical agents. For this purpose carbolic acid (from 25 per cent. to pure) or a strong solution of formalin may be used. Silver nitrate destroys the tetanus spores in laboratory experiments, but lacks penetration in the presence of albuminous matter. It is sometimes good practice to totally excise the wound, and even amputation must be considered in certain cases. The division of the umbilical cord and the treatment of the navel in the newborn must be done under the strictest asepsis. All wounds in which there is any suspicion of tetanus should be kept open and freely drained, and otherwise treated so as to discourage anaerobic conditions.

Tetanus spores gain entrance into wounds not only from manure, garden soil, street dust, and similar sources, but also from the hands, instruments, bandages, suture material, or other objects. It is important to remember that the tetanus spore is exceedingly resistant to heat and chemical agents, and that in surgical and obstetrical practice confidence should not be placed simply upon boiling to destroy the spores. Very particular care must be exercised in the disinfection of substances injected into the body, such as gelatin and other organic materials. For the destruction of tetanus spores complete confidence may be placed in the autoclave, in which a temperature of 120° C. for 20 minutes is attained, or exposure to dry heat at 160° C. for 1 hour.

It should be remembered that tetanus toxin is manufactured in the wound and is thence transported mainly along the nerve roots to the spinal cord and brain. It is therefore important to destroy or neutralize the toxin in the wound. For this purpose dry tetanus antitoxin may be dusted upon the wound. Formaldehyde, even in comparatively weak solutions, destroys the activity of tetanus toxin.

Specific Prophylaxis.—Tetanus antitoxin is a specific and trustworthy preventive. Its use, however, must be understood to achieve satisfactory results. The antitoxin must be administered before the advent of symptoms, for after the tetanus toxin has combined with the motor nerve cells in the central nervous system it can neither be displaced nor neutralized with antitoxin. In such cases the most that the antitoxin can do is to combine with and neutralize the free toxin and thus pre-

vent further damage. This in itself is quite worth while in the treatment of tetanus. At least 1,500 units of tetanus antitoxin should be given as a prophylactic dose.[1] It is important to remember that the tetanus antitoxin is eliminated or otherwise disposed of in the body in the course of 10 days or 2 weeks. Therefore, in cases in which the wound does not heal well, as a result of mixed infection, or for other reasons, it is desirable to repeat the injection. This may be done at intervals as long as the danger persists. Occasionally tetanus bacilli persist in the pus-infected tissues, and, when the injected antitoxin has been exhausted, there may occur a late development of tetanus. Rowan [2] reports a fatal case of tetanus in spite of the prophylactic use of 2,000 units of antitetanic serum, given 5 hours after the accident. In this case, however, the symptoms appeared 25 days later. The wound in this case was a compound fracture with a free discharge of rather foul-smelling pus. Instances in which 1,500 units of tetanus antitoxin have failed to prevent the development of tetanus in this country are rare. The few failures in France and Germany may be attributed to the fact that in those countries it is customary to use a smaller amount or a less potent serum than is used in this country.

Wounds produced by blank cartridges and other Fourth of July accidents should always be regarded as suspicious, and should be given careful local treatment, supplemented with a prophylactic injection of antitoxin. The prevention of tetanus complication of vaccine wounds consists in:

1. The use of a reliable vaccine which has been biologically tested in accordance with the federal act.

2. Proper methods of vaccination to avoid unnecessary scabs and anaerobic wound conditions.

3. Surgical asepsis of the operation and after-treatment.

Tetanus and other wound infections may be avoided, in those exposed to accidents, by cleanliness of body and clothing. A bath before a battle is a reasonable protection said to be adopted in the Japanese Army and Navy. The common experience of mankind teaches him that most wounds heal without tetanus, and that tetanus is, in fact, a relatively rare infection. The physician, however, is in no case justified in taking chances, and it is one of the duties of the medical profession to teach the public that it pays to thoroughly cleanse and care for wounds, however trivial, at once, and in accordance with modern methods.

[1] As soon as symptoms appear 20,000 units or more of tetanus antitoxin should be introduced directly into the circulation by intravenous injection; some antitoxin may also be injected into the nerves leading from the wound. In tetanus, as in diphtheria, time is the important element. A few units introduced early are worth more than thousands late.

[2] *Jour. A. M. A.*, XIV, No. 7, Feb. 12, 1910, p. 533.

CHAPTER II

DISEASES SPREAD LARGELY THROUGH THE ALVINE DISCHARGES

TYPHOID FEVER

Typhoid fever is a sanitary problem of first magnitude, especially in this country, where it is unduly prevalent. In the United States typhoid fever stands fourth on the list of mortality tables: tuberculosis comes first, then pneumonia, cancer, and typhoid fever. The average fatality from typhoid fever being nearly 10 per cent., it would, therefore, take still higher rank on the morbidity tables. In 1910 there were 25,000 deaths from typhoid fever in the United States, representing at least 250,000 cases.

Our general attitude toward typhoid fever is inconsistent; familiarity has bred a remarkable indifference to the disease. Every case of typhoid fever means a short circuit between the alvine discharges of one person and the mouth of another. The physician has a dual duty in the care of a case of typhoid fever: one is to assist the patient, the other is to protect the community. On the other hand, the people should learn the lesson that a case of typhoid fever should be regarded as seriously as a case of cholera. These two diseases present many features in common. Both are intestinal infections of bacterial nature; in both diseases the alvine discharges contain the microorganisms which reinfect another person when taken by the mouth. Both diseases prevail especially in hot weather, both diseases are peculiar to man, so that the patient is the fountainhead of each infection. Water, food, fingers, and flies play a similar rôle in both instances. In the case of cholera the dread of the disease is an important factor in keeping it out of the country or in preventing its spread when once introduced. By strange contrast, there is a remarkable indifference to typhoid fever. A wholesome fear of typhoid fever would materially assist the health authorities in combating what may be considered one of the greatest health problems of the age. From the standpoint of preventive medicine, it is proper to regard an outbreak of typhoid fever as a reproach to the sanitation and civilization of the community

in which it was contracted. When the matter is better understood health authorities will be held responsible for this and other preventable infections, just as some one is now held responsible for preventable accidents.

Much harm has been done by insisting that typhoid fever is infectious, but not contagious; it is both—that is, communicable.[1]

Typhoid fever occurs both in endemic and epidemic forms. It may truly be regarded as pandemic. Normally, typhoid fever is a warm weather disease. It recurs as an annual crop from July to October.[2] Epidemics caused by infected water occur especially in the early spring, late fall, or winter months. Milk outbreaks may occur at any time of the year. Autumnal typhoid in our cities is due partly to infection contracted at health resorts, and has, therefore, been called a vacation disease.

Typhoid fever is more prevalent in rural districts than in cities. In the United States there is more typhoid fever in the southern states than in the northern zone. The only explanation to account for this is the influence of temperature, rural conditions, and association with the negro. Typhoid fever is no respecter of rich or poor; it attacks those in robust health, all ages, both sexes.

Typhoid fever is a disease which ordinarily attacks the individual during the period of greatest economic value to the community. The economic loss, therefore, is appalling, and has been estimated to reach the sum of no less than $100,000,000 annually in the United States. Again, typhoid fever is an infection against which the individual alone cannot protect himself wholly without the aid of the community.

Prevalence.—Typhoid fever prevails more or less in all countries—the amount of the disease, however, varies greatly. It appears to be a disease of defective civilization, for those communities paying least attention to sanitation, as a rule, suffer most. In the United States there are comparatively few communities of 1,000 inhabitants or more which, during any period of twelve consecutive months within the last decade, have been entirely free from typhoid fever. According to the United States census report for 1900, the average typhoid death rate in the United States was 46.5 per 100,000 inhabitants. In 1908 the death toll from typhoid fever was no less than 35,000 in the United States. In other words, one person in about 200 in the United States contracted typhoid fever that year. It is estimated that in 1910-11 the number of deaths was reduced to about 25,000. The seriousness of these figures may be judged by estimating the probable number of cases of typhoid fever among persons handling the milk supply. Take, for instance, a city, as Washington, receiving its milk from a

[1] For distinction between these terms see page 317.
[2] In the southern hemisphere the typhoid season is during our winter.

thousand dairy farms. On the average there will be about four persons on each farm who in one way or another come in contact with the milk. That makes 4,000 persons among whom about 200 cases of typhoid may be expected to occur annually. No wonder that milk-borne outbreaks of typhoid fever are common occurrences.

The rate of prevalence of typhoid fever in the United States in comparison with the rates of many other countries is very high. Thus, the annual death rate from typhoid fever per 100,000 population for the period 1901-1905 was: in Scotland, 6.2; in Germany, 7.6; in England and Wales, 11.2; in Belgium, 16.8; in Austria (1901-1904), 19.9; in Hungary, 28.3; in Italy, 35.2; while the rate in the United States during the same period was about 46.5.

A comparison between the prevalence of typhoid fever in this country and abroad is impressive. The following ten European cities with a total population of about 15,000,000 have an average typhoid rate of 2.4 per 100,000 during the 10 years 1901-10:[1]

ANNUAL DEATH RATES FROM TYPHOID FEVER PER 100,000 POPULATION IN 10 EUROPEAN CITIES

	Average for 10 years, 1901–1910	Average for 5 years, 1901–1905	1906	1907	1908	1909	1910
Stockholm	1.7	3	2	2	1	5	1.8
Christiania	2.4	3	4	2	2	1.7	1.6
Munich	2.5	4	2	3	3	1.9	1.4
Edinburgh	2.9	8	3	3	2	1.2	.3
Vienna	3.7	4	5	3	4	2.8	3.8
Hamburg	3.7	4	4	3	4	3.3	4.1
Berlin	3.8	4	4	4	4	4.2	2.9
Dresden	4.2	4	7	2	6	4.2	2.2
Copenhagen	4.5	8	4	2	7	2.7	3.6
London	4.7	8	6	4	5	2.2	3.3

The following fifteen European cities with a population of about 9,000,000 had a typhoid death rate of 5.3 per 100,000 in 1909 and only 4.5 in 1910:

[1] These facts and the following instructive tables are taken from: "The Necessity of a Safe Water Supply in the Control of Typhoid Fever," by Allan J. McLaughlin, *U. S. Pub. Health Reports*, XXVII, 12, March 22, 1912.

ANNUAL DEATH RATES FROM TYPHOID FEVER PER 100,000 POPULATION IN 15 OTHER EUROPEAN CITIES

City	1909	1910
Frankfort	1.5	0.9
Antwerp	1.0	2.3
Bristol	2.8	2.1
Nuremberg	2.6	...
Birmingham	5.0	3.9
Belfast	5.2	3.9
Lyon	5.8	4.4
Leeds	7.2	3.8
Liverpool	8.4	3.9
Sheffield	9.4	3.0
Rotterdam	6.4	6.5
Amsterdam	3.8	6.7
Paris	8.4	5.6
Bradford	4.3	9.2
Leipzig	8.3	7.5
Total average rate	5.3	4.5

The following eight European cities with a total population of 7,500,000 had a typhoid death rate of 13.9 in 1909 and 15.6 in 1910. These rates would be considered low in America, but the European officials consider the persistence of such rates to be a reflection:

ANNUAL DEATH RATES FROM TYPHOID FEVER PER 100,000 POPULATION IN 8 OTHER EUROPEAN CITIES

City	1909	1910
Glasgow	12.5	6.4
Budapest	9.4	13.6
Brussels	7.4	16.1
Dublin	15.7	12.2
Manchester	13.9	10.3
Moscow	13.8	15.0
Warsaw	13.5	17.4
St. Petersburg	25.2	33.7
Total average	13.9	15.6

To recapitulate, in northern Europe the 33 principal cities, with an aggregate population of 31,500,000, had an average typhoid death rate per 100,000 population of 6.5 in 1909 and 1910. This includes such notorious typhoid centers as St. Petersburg, which had a rate of 33.7 in 1910. The rate in St. Petersburg is considered to be due to the water supply, which is partly filtered and partly raw Neva water.

Let us now compare these rates with typhoid fever in America:

ANNUAL DEATH RATES FROM TYPHOID FEVER PER 100,000 POPULATION IN 50 CITIES OF THE UNITED STATES HAVING MORE THAN 100,000 INHABITANTS

City	1909	1910
Birmingham, Ala.	59.7	49.5
Los Angeles, Cal.	16.1	14.2
Oakland, Cal.	11.2	16.5
San Francisco, Cal.	13.9	15.5
Denver, Colo.	24.1	27.5
Bridgeport, Conn.	9.0	4.9
New Haven, Conn.	20.5	17.9
Washington, D. C.	34.3	23.2
Atlanta, Ga.	50.6	50.1
Chicago, Ill.	12.6	13.7
Indianapolis, Ind.	22.3	28.5
Louisville, Ky.	45.0	31.7
New Orleans, La.	28.4	31.5
Baltimore, Md.	24.9	42.0
Boston, Mass.	13.8	11.3
Cambridge, Mass.	7.7	9.5
Fall River, Mass.	21.3	15.0
Lowell, Mass.	10.5	19.7
Worcester, Mass.	8.4	15.7
Detroit, Mich.	20.5	23.0
Grand Rapids, Mich.	17.2	28.3
Minneapolis, Minn.	21.0	58.7
St. Paul, Minn.	18.9	19.5
Kansas City, Mo.	29.3	54.4
St. Louis, Mo.	16.2	14.9
Omaha, Nebr.	36.8	86.7
Jersey City, N. J.	8.8	11.5
Newark, N. J.	11.9	13.1
Paterson, N. J.	9.7	7.1
Albany, N. Y.	19.0	14.0
Buffalo, N. Y.	23.8	20.4
New York, N. Y.	12.1	11.6
Rochester, N. Y.	9.4	13.7
Syracuse, N. Y.	11.2	28.2
Cincinnati, Ohio	13.3	8.8
Cleveland, Ohio	13.3	17.9
Columbus, Ohio	19.6	18.1
Dayton, Ohio	26.9	21.4
Toledo, Ohio	41.7	37.2
Portland, Oreg.	22.0	22.4
Philadelphia, Pa.	22.3	17.5
Pittsburgh, Pa.	24.6	27.8
Scranton, Pa.	16.4	16.9
Providence, R. I.	11.4	17.9
Memphis, Tenn.	48.8	27.4
Nashville, Tenn.	52.0	48.9
Richmond, Va.	24.1	21.9
Seattle, Wash.	23.8	14.2
Spokane, Wash.	43.2	45.4
Milwaukee, Wis.	21.4	45.7

These 50 registration cities in the United States have an aggregate population of over 20,000,000. The average typhoid death rate in these cities for 1910 was 25 per 100,000 inhabitants.

Unit of comparison	Aggregate population	Deaths per 100,000 from typhoid fever, 1910
33 principal European cities in Russia, Sweden, Norway, Austria-Hungary, Germany, Denmark, France, Belgium, Holland, England, Scotland, and Ireland	31,500,000	6.5
50 American cities of 100,000 inhabitants or over	20,250,000	25.0
Excess of deaths from typhoid fever in American cities		18.5

The excess of 18 deaths per 100,000 in the urban population alone shows that we have had, in the 50 cities mentioned above, at least 3,600 deaths, and probably 36,000 cases of typhoid fever, which were preventable and should never have occurred. For the whole United States the number of cases for each year readily preventable by methods within our grasp would probably reach 175,000, and the deaths so avoided would total about 16,000. In 1909 there were more cases of typhoid fever in the United States than there were cases of plague in India, in spite of the fact that India's population is two and one-half times that of the United States.

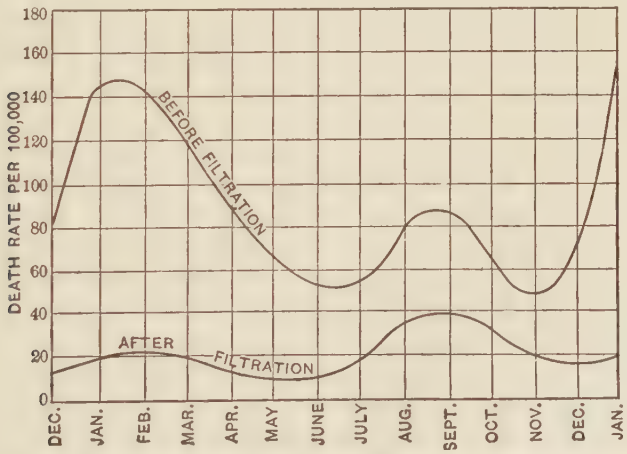

FIG. 10.—CURVE SHOWING DEATH RATE FROM TYPHOID FEVER IN ALBANY BEFORE AND AFTER FILTRATION OF WATER. (WHIPPLE.)

RESIDUAL OR "NORMAL" TYPHOID.—When a city such as Albany, Chicago, Lawrence, Lowell, or Pittsburg, which has been using grossly polluted water, is furnished with a water supply of good sanitary quality, there at once results a marked reduction in the amount of typhoid fever. The curve is not only lowered, but changed in character (Fig. 10—Albany). The remaining typhoid after the water-

borne infection has been removed is known as residual typhoid, and the curve in such cases is spoken of as the "normal" typhoid curve. The normal curve shows a distinct summer prevalence recurring with marked regularity each year, and lacks the great irregularities which characterize the curve of a community drinking badly infected water. Normal typhoid is endemic typhoid; Sedgwick has proposed the name "prosodemic" (*proso,* through, and *demos,* the people) as more expressive of this type of the disease. The amount of residual typhoid varies markedly in different localities; thus it is twice as high in the southern as in the northern part of our country; it is much greater here than in most parts of Europe.

Channels of Entrance and Exit.—The typhoid bacillus probably always enters by the mouth. Typhoid fever is generally regarded as primarily a gastrointestinal infection, although the disease itself is not produced unless the blood, glands, and other structures of the body are invaded with the specific microorganism. The typhoid bacillus grows and multiplies in the intestinal tract, penetrates the mucosa, and thus invades the body. The bacillus leaves the body mainly in the feces and urine, occasionally in the sputum and other discharges. Typhoid bacilli appear in the feces early in the disease; sometimes before the fever. Later in the disease they diminish in number and usually disappear during convalescence, although they may continue indefinitely (see "Bacillus Carriers," page 83). The feces may contain only a few typhoid bacilli; usually they are present in considerable numbers; occasionally they occur almost in pure culture, practically replacing the colon bacillus.

Typhoid bacilli commonly appear in the urine about the second, third, or fourth week. They grow well in this fluid both within and without the body, and may be present in such enormous numbers that the urine resembles a 24-hour-old bouillon culture. From the standpoint of prevention, it is very important not to neglect the virus in the urine. Urotropin (hexamethylenamin) in ten-grain doses or more three times a day diminishes the frequency of typhoid bacilluria, and is also effective in curing this condition when once established.

The sputum ordinarily does not contain the bacilli unless there is a pneumonia or severe bronchitis. The bacilli may be eliminated with the discharges from abscesses, such as periostitis, months and even years after the disease.

Diagnosis.—An early diagnosis of typhoid fever is important not only for the successful treatment of the patient, but is of vital importance in controlling the spread of the infection. The early diagnosis can only be assured through laboratory methods. Typhoid bacilli may be isolated either from the blood or the feces.

Blood Cultures.—Probably the easiest method, as well as the one

giving the maximum information, is through blood cultures. The taking of a little blood for this purpose is no more difficult or annoying to the patient than swabbing the throat for diphtheria. A few drops of blood may be obtained by puncturing the lobe of the ear or the finger, with the usual precautions to prevent bacterial contamination. A much better method, however, consists in withdrawing 5 to 10 c. c. of blood by means of a syringe from one of the veins at the bend of the elbow. The technique is very simple, and, if the needle is sharp, the patient scarcely feels the puncture. In fact, if the attention of the patient is distracted a skillful operator can withdraw 10 c. c. of blood in this way before the patient is aware that anything has been done. The blood may be planted in bouillon, or, better, in bile. After 24 hours in the incubator, any growth that occurs is transplanted to other media, a pure culture obtained, and tested for agglutination. Usually a pure culture is obtained in the first medium, so that the diagnosis may be established in 24 hours—at most, 2 or 3 days.

Typhoid bacilli appear in the blood early in the disease, perhaps occasionally during the prodromal symptoms. Kayser obtained positive results from 90 per cent. in the first week, 65 per cent. in the second, 42 per cent. in the third, 35 per cent. in the fourth. Our results in Washington were approximately the same. The typhoid bacilli probably do not grow in the blood during life. Their presence in the blood stream represents an overflow from the spleen and lymphatic tissues. The presence of typhoid bacilli in the blood may be taken to mean typhoid fever. The same cannot always be said if found in the feces or urine.

The Feces.—From the feces or urine typhoid bacilli are best isolated upon Endo's medium. This consists of a 4 per cent. alkaline agar containing fuchsin, which has been decolorized with sodium sulphite. Upon the surface of this medium typhoid colonies appear in 24 hours as translucent, dewdrop-like colonies, whereas colon bacilli and other organisms that produce acid and split the fuchsin appear as red colonies. Suspicious colonies are fished and may be tested at once under the microscope for agglutination, or may be planted in bouillon to obtain a growth sufficient for macroscopic agglutination tests. In any critical case pure cultures should be obtained and studied for morphological, cultural, and other biological characters. A modified Endo's medium and a rapid technique for diagnostic purposes, described by Kendall and used with success in my laboratory, are summarized as follows:

Technique.—Make plain, nutrient, sugar-free agar as follows: Tap water (cold), one thousand cubic centimeters; powdered agar, fifteen grams; peptone (Witte), ten grams; meat extract (Liebig), three grams. Cook in double boiler one hour. Make the reaction just al-

kaline to litmus by the cautious addition of NaOH. Cook fifteen minutes to set the reaction, and then filter through absorbent cotton.

The tap water should be as cold as possible and the agar should be "dusted" on the surface and allowed to settle into the medium before heat is applied and before the other ingredients are added.

After filtration, the medium is stored in flasks containing known amounts, conveniently in one hundred-cubic-centimeter lots, and sterilized in the autoclave.

To use the medium: (a) Prepare a ten per cent. solution of fuchsin in ninety-six per cent. alcohol. (b) Prepare a ten per cent. solution of sodium sulphite in water.

Add one cubic centimeter of (a) to ten cubic centimeters of (b) and heat in the Arnold sterilizer for twenty minutes=(c).

Add one per cent. of lactose (which must be chemically pure) to the agar medium described above, and heat in the Arnold sterilizer until the medium is melted and the lactose thoroughly distributed in it. The decolorized fuchsin solution (c) is then added in the proportion of one cubic centimeter of the mixture to each one hundred cubic centimeters of medium; then thoroughly mixed.

Plates are then poured and allowed to harden (with the covers removed) in the incubator for thirty minutes, after which time they are ready for inoculation.

Preparation of Feces for Inoculation.—The feces are collected preferably in the small rectal tubes described by Kendall.[1] A small portion of feces (about a loopful) is thoroughly emulsified in ten cubic centimeters of sugar-free broth, and preferably incubated one hour at 37° C. prior to the inoculation of the plates. This preliminary incubation does two things: the clumps of bacteria settle down, leaving a more uniform suspension of bacteria in the supernatant fluid for inoculation, and the bacteria undergo a slight development in a medium particularly suited for their growth. The thin suspension of the stool is now rubbed upon the surface of the agar plates by means of a bent, sterile, glass rod, and the plates incubated for 18 hours at 37° C. The suspicious translucent, colorless colonies are removed entire to small test-tubes containing one cubic centimeter of broth and incubated for two hours at 37° C. At the end of this time there will be sufficient growth to make the customary microscopic agglutination tests. Confirmatory cultural characters may be obtained by inoculating suitable media from the same tubes as those from which the organisms for agglutination were obtained.

Physicians should encourage boards of health to furnish diagnostic aids of a laboratory nature. Such work should be in the hands of

[1] *Boston Med. and Surg. Jour.*, CLXIV, No. 1, Sept., 1911.

specialists rather than entrusted to those who make occasional analyses. Early and accurate diagnosis is just as important to prevent the spread of other communicable diseases as it is with typhoid. These facts emphasized here will not be repeated under each disease.

Bacillus Carriers.—In about 4 per cent. of all cases of typhoid fever the patient continues to shed typhoid bacilli in the urine or feces during and after convalescence. Some persons shed typhoid bacilli without a clinical history of having had the disease. We therefore recognize three kinds of carriers: acute, chronic, and temporary. An acute typhoid bacillus carrier continues to discharge the infection not longer than 6 weeks following convalescence. A chronic carrier continues to discharge the bacilli 6 weeks or longer. A temporary carrier is a person who has not had clinical typhoid fever but who discharges typhoid bacilli for a short period. Albert states that 25 per cent. of all chronic typhoid carriers have never had typhoid fever; and further estimates that one in every 1,000 of the general population is a carrier.

While it would seem that typhoid bacilluria should be especially dangerous, a study of the cases indicates that most of the outbreaks that have been traced have been due to carriers who discharge the organisms in their feces rather than in the urine. It seems that typhoid carriers are more dangerous in certain seasons. More cases are traced to women[1] than to men. This is probably owing to the fact that the chief danger lies in handling foodstuffs, so that a carrier occupied as a cook or waitress is a special menace.

The question of preventing the spread of the disease through bacillus carriers is important and difficult. Surgical methods fail to cure carriers, for the typhoid bacillus may continue to grow in other parts of the intestinal tract than the gall bladder. Medical measures, such as urotropin, are efficient for bacilluria, but are of no avail in the fecal carriers. Attempts have been made to relieve the condition by the use of bacterial vaccines. Petruschky[2] and also Meader have reported encouraging results, especially with the use of autogenous cultures. So far certain cases resist all attempts to relieve the condition. It is unnecessary to place bacillus carriers *incommunicado*. It is sufficient to restrict their activities so that they may neither infect food nor their surroundings. With proper care and cleanliness typhoid carriers may present little danger to their fellow men. The problem, at present, is to detect the carriers, so as to establish a sanitary isolation, if not an actual quarantine.[3]

Resistance of the Virus.—The typhoid bacillus has no spore. It is, therefore, comparatively easy to destroy. The only difficulty present-

[1] Women are more subject to gall-stones.

[2] *Deut. med. Wochschr.*, July 11, 1912, XXXVIII, 28.

[3] The facts covering the infectivity of carriers are summed up by Ledingham, 39th An. Report Local Gov. Board, 1909-10, Supplement, p. 249.

ing itself is getting at the bacillus when imbedded in fecal masses. When dry, most typhoid bacilli die in a few hours; occasionally a few survive for months. The fact that typhoid bacilli are killed by drying renders infection through dust unlikely.

When a moist medium, such as water, milk, or urine, is heated to 60° C., practically all the typhoid bacilli such a medium may contain are killed. An exposure at 60° C. for 20 minutes will surely kill all of these microorganisms. They are not destroyed by freezing (see "Relation to Ice," pages 837 *et seq.*

In their resistance to germicides typhoid bacilli behave like the average non-spore-bearing bacilli. Thus bichlorid of mercury, 1-1,000, phenol, 2½ per cent., formaldehyde, 10 per cent., are effective upon the naked germs. In order to kill the typhoid bacilli in feces special precautions or stronger solutions are necessary (see page 1030).

The viability of typhoid bacilli in feces is very variable, depending on the composition of the feces and the varieties of other bacteria present. Sometimes the typhoid bacilli in feces perish in a few hours, usually in a day; under exceptional circumstances they may live for much longer periods. In the Plymouth epidemic typhoid bacilli probably remained alive and virulent in the feces, exposed to the winter's cold, for several months. Levy and Kayser found they remained alive in feces for 5 months in the winter. The life of the organism in privies and in water is usually comparatively short. In nature they die, as a rule, in water in about 7 days and often after 48 hours. They probably live longer in clean water than in contaminated water. In the outer world symbiosis plays an important part, also the presence of deleterious chemicals, temperature, light, desiccation, dryness, and other factors known to be injurious to spore-free bacteria. As a rule, the typhoid bacillus does not survive long in the soil under the usual conditions.

Typhoid Bacillus in Nature.—The typhoid bacillus should be regarded as a pathogen, not as a saprophyte. It lives and grows principally in the human body. It has a tendency to die in water, air, soil, upon fomites, or in nature generally. The grand exception to this statement is in the case of milk, in which the typhoid bacillus grows well.

The typhoid bacillus is much more widely distributed in man than the cases indicate. Thus, in the District of Columbia, of 1,000 healthy persons examined during the typhoid season of 1908, typhoid bacillus was found in the feces in 3 instances. At least one and perhaps two of these individuals were regarded as temporary carriers. In each instance the organisms were found only once. The population of the District of Columbia in 1908 was 300,000, and at the ratio of 1 per 1,000 this would represent about 300 healthy persons in that community har-

TYPHOID FEVER: 1902 TO 1906

DEATH RATE PER 100,000 OF POPULATION

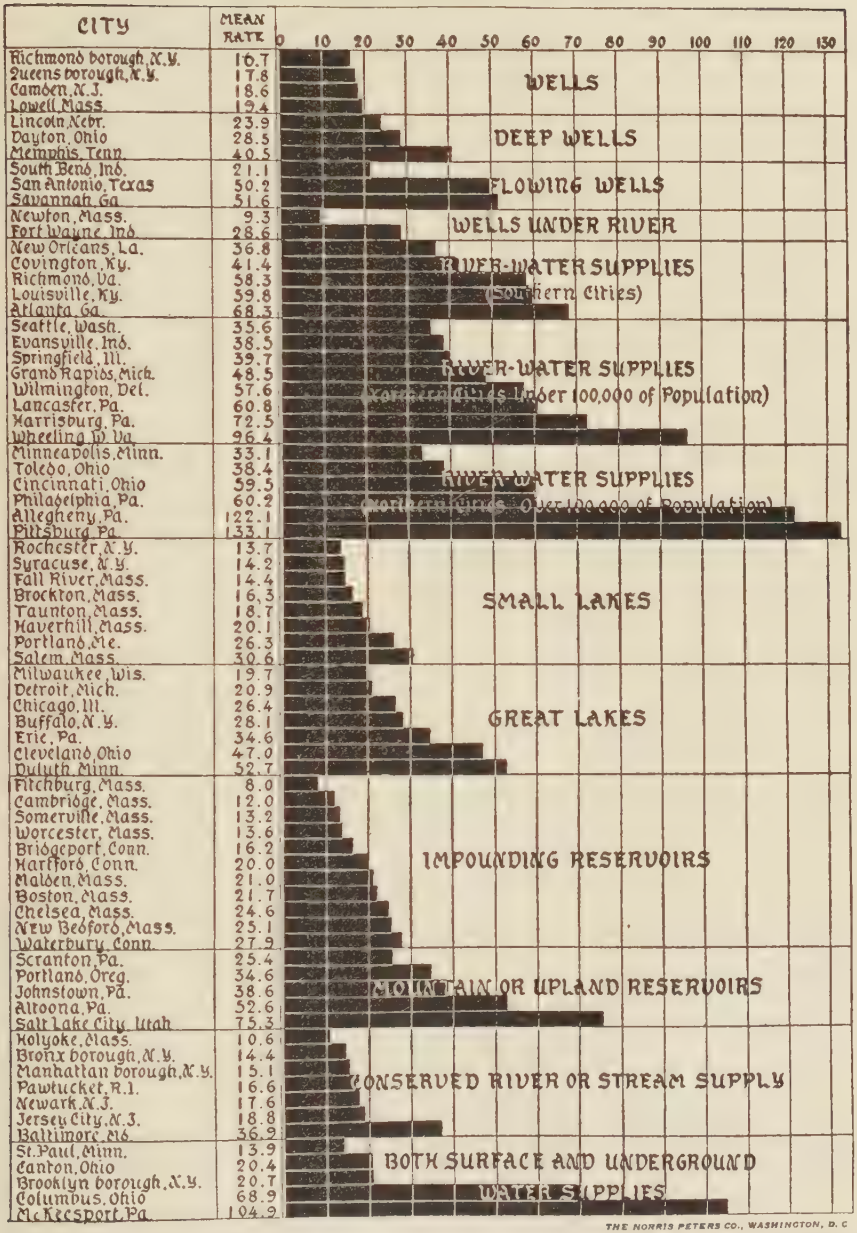

FIG. 11.—INFLUENCE OF PUBLIC WATER SUPPLIES ON THE TYPHOID FEVER DEATH RATE. (Diagram prepared by Marshall O. Leighton, U. S. Geological Survey, from figures furnished by Dr. Cressy L. Wilbur, Chief Statistician of Vital Statistics, Bureau of the Census—from Kober.)

boring and shedding typhoid bacilli for a brief period of time during the typhoid season.

Modes of Spread.—Typhoid fever is spread either by direct or indirect contact—indirectly through water, milk, and other foods; through "contacts," and also flies, fingers, and fomites. Each of these modes of spread needs separate consideration.

Water.—Water-borne typhoid is a common occurrence. Not long ago it was regarded as the sole or usual mode of spread; now we know that this was a mistake. Most fecal matter ultimately finds its way to water; most water courses draining inhabited regions are contaminated with human feces. Surface water is, therefore, apt to contain typhoid bacilli. The fact that there may be no clinical case of typhoid fever in the drainage area is no guarantee that the water may not be infected—in view of the prevalence of missed cases and bacillus carriers.

Fortunately, typhoid bacilli do not grow and multiply in water under natural conditions. They usually die in a few days, and rarely persist longer than 7 days. They succumb more quickly in some waters than others, more quickly in summer than winter. Thus Reudiger[1] has shown that typhoid bacilli die less quickly in the Red Lake River in Minnesota when exposed in dialyzing membranes in the river with ice than in the open river. He also showed that colon bacilli as well as typhoid bacilli disappear much more rapidly from polluted water during the summer months than during the winter months when the river is protected with a covering of ice and snow. Reudiger considers that the destruction of the typhoid bacillus in river water during the summer months is in a large measure due to the growth of microscopic plants, and other organisms which give off dialyzable substances which are harmful to *B. typhosus*. One of the reasons for believing in the existence of such poisons in water is the fact that typhoid cultures in a collodion sac placed in water die more quickly than otherwise. Further, the effect of the direct rays of the sun are entirely lost when the river is covered with ice and snow.

Water plays a large but diminishing rôle in the spread of the typhoid bacillus. The great water-borne epidemics have overshadowed the other media of communication. We know that the larger part of the typhoid now prevalent in this country is not water-borne; Whipple in 1908 estimated it at 35 per cent.; it is now no doubt much less. Typhoid fever may be excessively prevalent, even epidemic, in a city having a water supply of good sanitary quality.

In the vast majority of cases water-borne typhoid is contracted from a surface supply, that is, a river, small stream, pond, or lake.

[1] *Jour. Am. Pub. Health Ass.*, June, 1911, Vol. I, No. 16, p. 411.

Ground water becomes a source of danger only under special conditions (see chapter on water).

Water-borne epidemics present certain definite characteristics. They almost always occur in the spring, fall, or winter, when the water is cold. Most of the great water-borne epidemics have occurred in northern cities, both in this country and in Europe. They usually have a sharp onset, the curve rises to a peak, and declines rapidly. The pollution is usually nearby; that is, there is a rather direct transfer of fresh virulent infection. Granting that the typhoid bacillus does not grow in cold water, there must be a very considerable dilution in most of the epidemics.

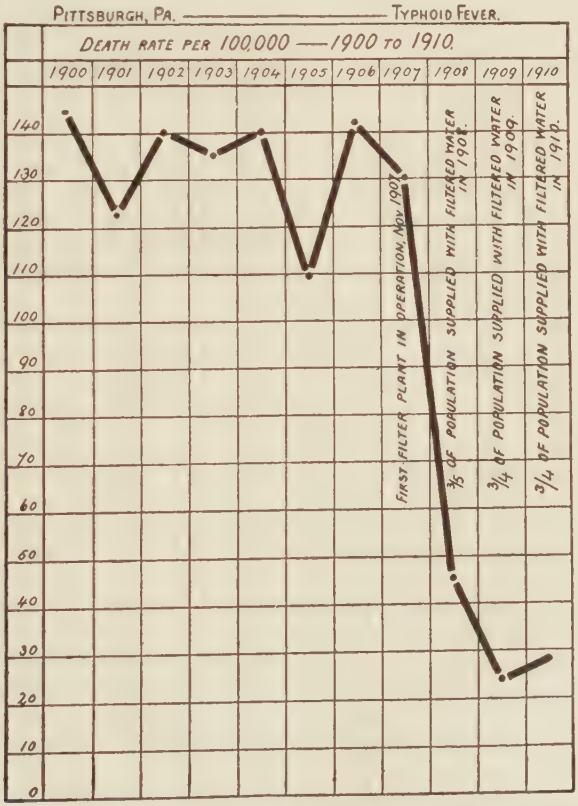

FIG. 12.—IMMEDIATE AND STRIKING EFFECT OF PURIFYING A BADLY INFECTED WATER SUPPLY UPON THE TYPHOID SITUATION.

The following examples are given of the fact that water-borne outbreaks of typhoid fever occur during the winter, fall, or early spring, when the water is cold. Thus we have the water-borne epidemic in Plymouth, Penn., in 1885, which began with the spring thaw and was doubtless produced from the frozen accumulation of typhoid ex-

crement from a single case. Very similar to the Plymouth outbreak was that at New Haven, Conn., in 1901. The outbreak at Ithaca, N. Y., started in epidemic proportions in January. The epidemic in Sherbourne, England, in 1873, likewise started in January. Four acute epidemic exacerbations are recorded in Philadelphia in December of the years 1884, 1890, 1899, and 1903. Several similar epidemics have occurred in the winter time in Chicago—one in January, 1890, another in January, 1896, and one in March, 1891. Another striking instance is the epidemic in Newark, N. J., in February, 1899, and one in December, 1891. Abroad, epidemics are recorded in Berlin in February, 1899, in Paris in February, 1894, and in Vienna in November, 1888. All of these are generally believed to have been water-borne and must have taken place when the water was very cold. In fact, as previously pointed out, extensive water-borne epidemics of typhoid fever rarely occur in the summer time.

The epidemiology of water-borne typhoid caused by distant, diluted and attenuated infection is not understood. It was formerly thought that a high typhoid rate necessarily meant badly infected water. We know now that this does not necessarily follow, as has been proven by the experiences in Washington, Winnipeg, army camps, and many southern cities.

Almost all the water-borne epidemics of typhoid fever rest upon circumstantial evidence. It is difficult to isolate the typhoid bacillus from water, and the damage is usually done before suspicion points to the water.[1]

It is clear that in cities which have had safe water supplies for a period of years the rate should not be above 5 per 100,000, unless some unusual condition exists, such as poor control of milk or lack of control over patients and carriers, and disregard of modern sanitary knowledge.

No single measure in reducing typhoid fever on a large scale approaches the effect of substituting a safe for a polluted water supply. As an instance of this wholesale saving of human life, the reduction of typhoid fever in four American cities is shown in Fig. 13, p. 89.

Ice.—Ice may, under exceptional circumstances, occasionally be the vehicle by which typhoid bacilli are transferred. Freezing does not kill *B. typhosus,* but there is a great quantitative reduction not only in the act of freezing, but during storage, hence the danger is greatly lessened. The most suggestive outbreak of typhoid fever attributed to ice was reported by Hutchins and Wheeler in 1903 in the St. Lawrence Hospital, three miles below Ogdensburg. A few other instances in which ice is believed to have conveyed the infection have been re-

[1] Examples of water-borne outbreaks of typhoid fever will be found in the chapter on water.

ported, but are based upon flimsy evidence. The fact that natural ice is usually stored many weeks or months before it is used is a sanitary safeguard. Manufactured ice made from distilled water and handled with cleanly methods is above reproach. For a discussion of ice in relation to typhoid fever and other infections see page 840.

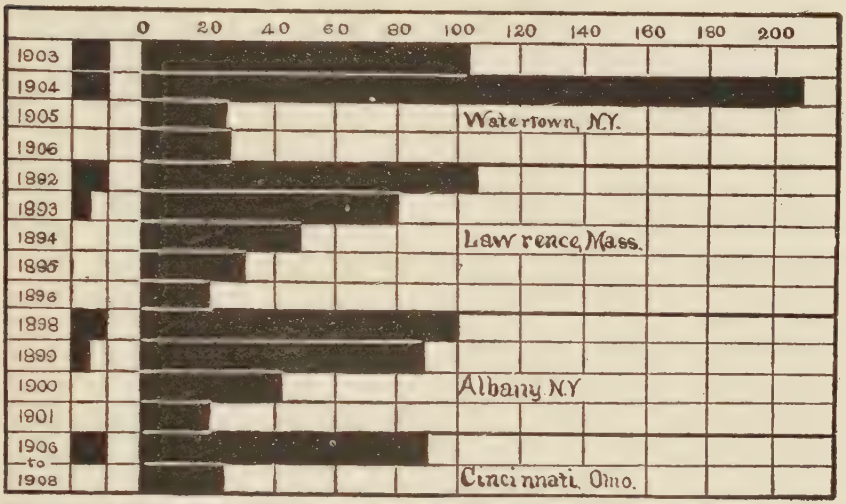

FIG. 13.—ABRUPT REDUCTION IN DEATH RATES FROM TYPHOID FEVER INCIDENT TO WATER PURIFICATION IN FOUR AMERICAN CITIES.

MILK.—Trask collected 317 typhoid epidemics up to 1908 caused by infected milk. Since then many more instances have come to light. Doubtless many milk outbreaks have escaped attention or have been attributed to water or other sources. The typhoid bacillus grows well in milk, and it is now realized that this medium is a frequent and important mode of communication. Most milk outbreaks are reported either in England or America. On account of the almost universal custom of boiling the milk in European and tropical countries, milk outbreaks are rarely reported from these regions. During the four years' study of typhoid fever in Washington, it was found that at least 10 per cent. of the cases were milk-borne.

The milk usually becomes contaminated on the farm, from a case or a carrier. It may also become infected in transportation, at the city dairy, or in the home. Milk outbreaks come abruptly, rise to a peak like a water epidemic, and subside rather sharply. There are comparatively few secondary cases. Milk-borne epidemics of typhoid fever have certain characteristics which permit ready recognition.

(a) There is a special incidence of the disease on the track of the implicated milk supply. The outbreak is localized to such areas.

(b) The better class of houses are invaded, and persons in better circumstances generally suffer most.

(c) Those who drink milk are chiefly affected and those suffer most who are large consumers of raw milk.

(d) The incidence is high among women and children.

(e) The incubation period is shortened perhaps on account of the large amount of infection taken.

(f) More than one case occurs simultaneously in a house. This is a very suspicious circumstance to the epidemiologists. The first indication of a milk outbreak in a city with a good water supply is usually the fact that two or more persons in a household came down with typhoid fever within a few days of each other.

(g) Clinically the disease usually runs a mild course, owing to the fact, no doubt, that the virus becomes attenuated in the process of multiplication in the milk. In water-borne typhoid the same germs are ingested that were passed; in milk-borne typhoid it is the succeeding generations that are ingested.

Milk-borne outbreaks are sometimes very extensive. One of the largest epidemics occurred in Boston (Jamaica Plain) in March and April, 1908. Four hundred and ten cases were reported; 348 of them drank the suspected milk. Among the first victims of the disease was the milkman, who was believed to have infected the milk through tasting it. The number of persons involved in a milk-borne epidemic varies greatly, depending upon the amount of milk infected and other factors. It must not be uncommon for a single bottle of milk or a small quantity to become infected, and thus transmit the disease to one or two persons. Such instances are exceedingly difficult to trace. Oftttimes the milk becomes infected from a carrier. An instance of this occurred in Washington (Georgetown) in 1908. In this case the milkmaid had typhoid fever 18 years previously. Examinations showed almost pure culture of *B. typhosus* in her feces. Fifty-five persons who drank the infected milk contracted the disease.

Milk Products.—Fresh milk products, such as cream, ice-cream, butter, and buttermilk, and fresh cheese, may contain the typhoid bacillus, and are occasionally media of communication.

Cream contains more bacteria than the milk from which it is taken. The use of infected cream in coffee, on cereals, etc., is sufficient to cause the disease. Several instances in the Washington studies were traced to such use of cream. As a rule, coffee in the cup is not hot enough to kill the typhoid bacillus.

Freezing kills only a certain percentage of the typhoid bacilli. In Washington several cases of the disease were traced to ice-cream.

Bruck has shown that the typhoid bacillus will live in butter for 27 days.

Buttermilk may be quite as dangerous as the cream from which it is derived. In cheese the time of fermentation, symbiosis, etc., les-

sens the likelihood of survival of the typhoid bacillus. Fresh cream-cheese, such as Cottage cheese, may be responsible for an occasional case.

Oysters, Mussels, and Shellfish.—The first outbreak of typhoid fever attributed to this source was investigated by Conn at Wesleyan University, Middletown, October, 1894. Twenty-five cases were attributed to eating infected oysters; 4 died. Not all of those who took sick had clinical typhoid fever. Some had gastrointestinal disturbances with illness lasting but a few days. About one-quarter of those attending the dinners at which the oysters were served were made ill.

A similar instance occurred at the Mayors' banquets at South Hampton and Winchester, in 1903.

In the Washington studies it seems that oysters and shellfish play a minor rôle in the spread of the disease, which occurs mostly in the summer time, while oysters and similar sea food are relished mainly in winter. Comparatively few of the cases studied gave a history of having eaten oysters within 30 days prior to the onset of the disease. Oysters become especially dangerous when consumed soon after taking them from a polluted bed, or when floated or bloated in infected water. (For further discussion of this topic, see page 566.)

Fruits and Vegetables.—Vegetables, such as celery, lettuce, and radishes, partaken of raw, and grown on land fertilized with fresh night soil, may be dangerous, and this probably accounts for an occasional case. In large cities it is practically impossible to trace this source of infection. It therefore remains more a suspicion than a conviction. In Hackney, London, two local outbreaks were traced to watercress taken from a polluted stream. In Springfield, Mass., an outbreak which occurred in the summer of 1905 was attributed to infected fruits and vegetables.

Creel[1] found typhoid bacillus upon the tips of leaves of plants cultivated in contaminated soil. Under conditions most unfavorable to the *B. typhosus* the infection lasted at least 31 days—a period sufficiently long for some varieties of lettuce and radishes to mature.

Flies.—The evidence is now complete that the common house fly (*Musca domestica*) may convey the infection of typhoid. It is not inappropriately called the typhoid fly. The typhoid bacilli may be smeared upon the feet or other parts of the insect, or may live in the intestinal tract and pass in the dejecta in almost pure culture. Flies live, feed, and breed in fecal matter and decomposing organic substances of all kinds. It is easy to see how they may convey infections from this source to our food, lips, or fingers. Alice Hamilton isolated typhoid bacilli from 5 out of 18 house flies captured in Chicago in the privy and on a fence near a sick room. It has been shown experimentally that

[1] *Public Health Reports*, Feb. 9, 1912, p. 187, XXVII, 6.

living typhoid bacilli may remain upon the bodies of flies for as long as 23 days. Special attention to the rôle played by the fly was given by Reed, Vaughan, and Shakespeare in their studies of the prevalence of typhoid fever in our army camps in 1898. They concluded that flies undoubtedly served as carriers of the infection and attributed about 15 per cent. of the cases to this mode of communication. They found that "flies swarm over infected fecal matter in the pits and then deposit it and feed upon the food prepared for the soldiers at the mess tents. In some instances, where lime had recently been sprinkled over the contents of the pits, flies with their feet whitened with lime were seen walking over the food." The danger from fly transmission varies very much, and depends upon circumstances. In a camp it is considerable; in a well sewered city the risk is diminished. In our Washington studies we could find no relation between fly abundance in the summer of 1908 and typhoid prevalence. It is not possible to express mathematically the percentage of cases caused by flies—the figures would vary greatly, depending upon circumstances. The danger of typhoid from flies in cities has doubtless been overstated. However, if only one per cent. of the cases were thus transmitted, the suppression of flies would still be quite worth while (page 223).

Dust.—Typhoid bacilli soon die when dried, especially when exposed to the sun and air. Dust-borne infection in this disease must be rare. In the South African war there were frequent dust storms in some localities, so that the food was covered with dust and sand. Some of the infection was believed to have been conveyed in this way.

Fomites.—The infection may be conveyed upon soiled linen, blankets, and other objects. It was believed by Reed, Vaughan, and Shakespeare that the clothing, blankets, and tents in the Spanish-American war became infected and were a prime factor in spreading the disease. After the South African war some of the blankets used by the troops were sent back to England and used on a training ship, on which typhoid fever appeared. The blankets were found to be dirty and soiled with fecal matter, from which Klein is reported to have obtained living typhoid bacilli. The danger of fomites contaminated with fresh infection is real, and emphasizes the importance of disinfecting bedding, towels, and other fabrics.

Soil.—The soil, long regarded as the most important factor in the spread of typhoid fever, and by Pettenkofer and others considered an essential element, is now given scant consideration. Pollution of the soil, however, cannot be disregarded. The typhoid bacillus may live for a long time in sewage-soaked earth. A surcharged soil may endanger the water, milk, and other foods, or infect through flies and other means (see Soil).

Contact Infection.—"Contact" is a convenient term to indicate

the spread of infection directly or indirectly as a result of close association between the sick and the sound. Actual contact is not necessarily implied. The term is used to indicate the transfer of the infection through a short intervening space in a brief period of time (see page 314). Thus the infection may be passed from one to another through kissing, soiled hands, remnants of food, infected thermometers, or tongue depressors, contaminated towels or other fabrics, cups, spoons, glasses, etc. If the nurse infects a cup of milk or glass of water that carries the infection to another member of the household, such cases are included under "contacts." The infection may also be spread in the household by flies, fingers, and various other means, usually difficult to trace, and which are, therefore, all included under this group. Regarded in this light, contacts play a major rôle in the spread of the disease.

Extensive municipal outbreaks have been reported as largely or entirely due to contact infection. Winslow in 1901 studied such an outbreak in Newport. Others have been reported from Knoxville, Winnipeg, Springfield, and from Germany and England. Koch regarded the spread of typhoid in Trier in the light of contact infection. Freeman says that the majority of outbreaks in the smaller towns of Virginia are due to this cause. Extensive outbreaks in institutions are often due to contact with mild cases or carriers. Flies, fingers, and food (Sedgwick), and dirt, diarrhea, and dinner (Chapin), which too often get sadly confused, explain the occurrence of many a case of contact infection in typhoid fever and other infections.

In army camps with clean water and good milk, contact infection may rise to epidemic proportions. In the Spanish-American war, of 107,000 of our troops in camp, 20,000 contracted typhoid, mostly by "contact." Similar conditions prevail in rapidly growing cities, in crowded apartments, and congested regions with a susceptible population and other favoring conditions. The danger of contact is well shown by the frequency with which nurses, ward attendants, house physicians, and others similarly exposed take typhoid fever. Studies of the incidence of the disease in the Massachusetts General Hospital, Boston, in the Presbyterian Hospital, Philadelphia, and the Johns Hopkins Hospital, Baltimore, show that typhoid fever is at least twice and may be 8 times as prevalent among those who come in close and frequent association with the patient as among the population at large. Further, the disease contracted under such conditions seems to run a course of more than ordinary severity, with a greater number of complications and with a high mortality. This is doubtless due largely to the fact that the contactors receive fresh virulent virus.

In our studies of typhoid fever in Washington we were impressed with the importance and frequency of contact infection in that en-

demic center. In 1907 we attributed 6 per cent. of the cases to contacts; in 1908, 15 per cent., and in 1909, 17 per cent. This included only contact with cases during the febrile stage of the disease. In Strassburg, Kayser attributed 16.8 per cent. of the cases occurring during 3 years in that city to contact infection. Little groups of 4, 6, to 12 or more cases following a primary case in a suburban focus, in my experience, frequently fall in the category of contacts.

According to Conradi, the infection is transmissible most often during the early stages of the disease, sometimes even during the period of incubation. The Washington studies do not support this view, for we found the disease is communicated during all stages, and especially during convalescence. This may be due to the fact that during this time the patient moves about and scatters the infection over a wider radius.

Typhoid fever, in view of all the facts, must now be regarded as a "contagious" disease. We will never have an end of it until it is so regarded and treated accordingly.

Preventive Typhoid Inoculations.—An active immunity to typhoid fever may be artificially induced by introducing dead typhoid bacilli into the subcutaneous tissue. Living cultures or bacillary extracts may also be used. The procedure is harmless, rational, and effective.

Our knowledge of inoculations against typhoid fever began with the work of Pfeiffer and Kolle,[1] who inoculated two volunteers in 1896. About the same time Almroth Wright [2] inoculated several persons, and in 1898 continued the work upon an extensive scale in India upon 4,000 British soldiers. In 1900, during the Boer war, Wright, together with Leishman, prepared a vaccine [3] and supervised the inoculation of 100,000 British troops. The results in India were quite encouraging, but for various reasons the same procedure in South Africa was not as satisfactory as had been anticipated. Prophylactic inoculation on the advice of Koch was used by the Germans in the Herero campaign in southern West Africa in 1904. The prophylactic was voluntary and only about half of the command (7,287 men) availed themselves of it. The results, while good, fell short of expectations. In this country Richardson was the first to advocate and practice inoculations as a means of protection against typhoid fever. The best results have been obtained in the United States Army under the direction of Major Russell.

Leishman [4] in his Harben lecture (1910) explains the lack of suc-

[1] Pfeiffer and Kolle: *Deutsche med. Wochnschr.*, 1896, XXII, 735.

[2] Wright: *Lancet*, London, Sept. 19, 1896, 807; *Brit. Med. Jour.*, Jan. 30, 1897, 16.

[3] The material injected is called a vaccine and the process spoken of as vaccination. The term in this connection is a little confusing. Inoculation is better.

[4] Leishman, W. B.: *Jour. Roy. Inst. Pub. Health, London*, 1910, XVIII, 394.

cess in early years by saying that the vaccine may have been made less efficient by the use of too great heat in killing the bacilli. Further, it should be noted that smaller doses and fewer injections were given then than now.

The typhoid vaccines may be prepared in a number of different ways. Usually dead bacilli are used, although live bacilli have been inoculated. The bacilli may be killed either with the aid of heat or germicidal substances; the dead or live bacilli may be sensitized by the addition of antityphoid serum; the vaccines may be prepared with pulverized bacilli, from bacillary extracts, or by the use of various chemical methods.

Usually the vaccine is made from a twenty-four-hour-old culture killed by heating to 60° C. for one hour or less. Overheating probably impairs the immunizing power of the vaccine. Most typhoid bacilli die before the temperature reaches 60° C. Some of the strains have a lower thermal death point. Stone heats only to 53° C. for one hour, depending upon phenol (0.5 per cent.) to sterilize the culture. Cultures killed without heat have perhaps greater protective properties.

Certain cultures seem to cause the production of more antibodies than others. In the earlier work it was believed that the more virulent strains produce a greater protection. This is doubtful, for it appears that the protection afforded is not in proportion to the local or general reaction, but to the amount and variety of antibodies stimulated.

The injections are given subcutaneously at intervals of five days. From 50,000,000 to 100,000,000, sometimes 1,000,000,000, dead typhoid bacilli are injected at each inoculation. The number of inoculations varies with different authorities. At least 3, preferably 4, should be given; the greater the number of injections the greater the immunity induced.

A reaction at the site of the inoculation occurs in about 10 per cent. of persons. The reactions are usually moderate and never serious. They consist of local manifestations, of irritation, and inflammation about the site of inoculation, such as pain, redness, swelling, edema; also general symptoms, such as malaise, pains in the back and limbs, and fever. Children, as a rule, react less than adults. Of 1,101 persons inoculated by Hartsock, 11 per cent. showed no reaction, 83 per cent. mild reaction, 5 per cent. a moderate reaction, and 1 per cent. a severe reaction. All the cases had a slight local tenderness and redness at the point of inoculation. The symptoms of the reaction usually pass in 24 hours. The number and character of the reactions in the experience of the United States Army [1] are shown in the following table:

[1] Russell, F. F.: *Jour. A. M. A.*, LVIII, No. 18, May 4, 1912.

	Number of doses	Reaction, Absent	Reaction, Mild	Reaction, Moderate	Reaction, Severe
First dose	45,680	68.2%	28.9%	2.4%	0.3%
Second dose	44,321	71.3%	25.7%	2.6%	0.2%
Third dose	38,902	78.0%	20.3%	1.5%	0.1%

The best time to give the treatment is late in the afternoon, for then the severest part of the reaction is over by the morning. The injections are usually given into the subcutaneous tissue of the outer side of the arm or into the abdominal wall; sometimes the interscapular space.

There is no laboratory index of the degree or duration of the immunity produced as a result of the inoculations. The following antibodies appear in the blood: agglutinins, precipitins, opsonins, lysins, stimulins. There are factors involved in the immunity not understood, and, therefore, the presence or absence of typhoid fever among individuals protected in this manner is the only index of value.

The negative phase advanced by Wright and denied by Leishman and others probably does not occur. At least there appears to be no increased susceptibility to the disease during the so-called negative phase. There is, therefore, no known objection to giving the prophylactic to those exposed to the disease or during an epidemic. In fact, the vaccines have been used as a therapeutic agent during the illness.

The immunity varies in degree and also in duration; at least one year (Pfeiffer and Kolle's vaccine); four years (Wright's vaccine). On the average, the immunity may probably be depended upon for 2 or 3 years when produced by 4 injections of dead bacilli. The immunity may be prolonged or renewed by recourse to reinoculation. One attack of typhoid fever, however mild, produces, as a rule, a lasting immunity. Second attacks, however, occur. Draschfeld's figures, based on 2,000 persons in the Antwerp Hospital, show that only 0.7 per cent. of that number were affected twice.

The results of typhoid inoculations can no longer be questioned. The morbidity is lowered in those who have been properly "vaccinated"; the figures are too recent to state just how much. The most striking effect is in the lowering of the mortality. The latest summing up of the antityphoid inoculations is by Leishman in the July and September, 1910, numbers of the Journal of the Royal Institute of Public Health, xviii, Nos. 7, 8, and 9; also Report of the French Commission, Public Health Reports, P. H. & M. H. S., October 6, 1911, xxvi, 40, 1507.

The best results have been obtained in the United States Army,

where the vaccinations are done under the supervision of Major Russell.[1]

The health record established by the Maneuver Division of the United States Army at San Antonio, Texas, during the summer of 1911, is a triumph in preventive medicine. The division had a mean strength of 12,801 men. All were treated with the typhoid vaccines. The result was that from March 10th to July 10th only two cases of typhoid fever developed; no deaths. One patient was a private of the hospital corps who had not completed his immunization, having taken only two doses. His case was very mild and probably would have been overlooked but for the rule that blood cultures were made in all cases of fever of over 48 hours' duration. The other case was a teamster who had not been inoculated. Among the 12,801 men there were only 11 deaths from all diseases. Typhoid fever prevailed at the time in the neighborhood. Thus, there were 49 cases of typhoid fever with 19 deaths in the city of San Antonio during this period. This contrasts markedly with the typhoid record of the United States Army during the Spanish-American war, when the typhoid record of a division of volunteer troops camped at Jacksonville, Florida, in 1898, under conditions similar to those at San Antonio, was as follows: The division at Jacksonville had 2,693 cases with 248 deaths, which was about the average typhoid incidence of the camps. Since the year 1904, with an improved vaccine, more than 100,000 British troops have been inoculated without any untoward result. The protection afforded may be seen from the most recent figures from India, reported by Col. R. H. Firth.[2]

"In that period there were, in all India, 112 cases of typhoid, with six deaths, among the protected men, and forty-five cases with four deaths among the non-protected. The protected population was 63,624 persons, and the non-protected 8,481. From these data we find the case incidence per thousand among the protected to be 1.7 and among the non-protected to be 5.3. If we take the mortality and express it as per million, then the ratio for the protected is 94, and for the non-protected 471. That is to say, the incidence for typhoid for the first half year was roughly five times as great among the non-protected as among the protected."

Spooner reports that in the Massachusetts General Hospital, among the nurses and others exposed to typhoid fever, 80 per cent. of whom have been inoculated during the past three years, not a case has been contracted, and for the first year in the history of the institution there were no cases among the nurses or attendants. The case morbidity in training schools for nurses in Massachusetts during three years was nearly nine times greater in the uninoculated than among the inoculated.

[1] *Loc. cit.*, p. 95.
[2] Firth, R. H.: *Jour. Roy. Army Med. Corps*, London, 1911, XVII, 495.

Metchnikoff and Besredka[1] failed to protect chimpanzees against typhoid infection by means of killed bacilli, but obtained immunity apparently as definite as that produced by an attack of the disease by the use of living cultures.[2]

SUMMARY.—Preventive typhoid inoculations involve no risk whatever, and are especially applicable to those unduly exposed to the infection, such as nurses, hospital attendants, physicians, travelers, soldiers in camps, persons in epidemic localities, and persons in the family of a bacillus carrier. The method has been proposed for general use among the public in endemic foci, but it is a question whether this artificial method of acquiring immunity would serve as good a purpose in the end as fighting the disease along the lines of general sanitation —which has been so successfully done in many European centers. It would certainly be a mistake to immunize the population with this artificial method to the neglect of general sanitary improvements, such as good water, clean milk, fly suppression, cleanliness, and personal hygiene. The question as to whether the vaccinations may or may not increase the number of bacillus carriers should also be determined. Because a person has received the protection afforded by typhoid inoculations is no reason for reckless disregard of other prophylactic measures.

Management of a Case so as to Prevent Spread.—Success depends upon an early and accurate diagnosis. All cases of typhoid fever and all cases suspected of being typhoid fever should be isolated. This does not mean imprisonment in a lazaretto. The proper place to care for typhoid fever is in a suitable hospital. A private home is a poor makeshift for a hospital, and it is unreasonable to turn a household into a hospital for 4 to 8 weeks or longer. The room in which the patient is treated should be large and well ventilated, and should contain no unnecessary furniture, curtains, carpets, etc. It must be kept scrupulously clean, dry sweeping and dusting prohibited; and well screened.

The case should be reported to the health authorities without delay, and the house should be placarded so as to warn others, and visiting discouraged. Under no circumstances should visitors be admitted into the sick room.

The disinfection of the stools, urine, sputum, and other excretions is of the first importance, and should be carried out with great care and conscientiousness. For the urine, sufficient bichlorid may be added to make a 1-1,000 solution, or carbolic, 2.5 per cent., formalin, 10 per cent., and allowed to stand one hour before discarding. Stools may be disinfected with bleaching powder, 3 per cent.; milk of lime (1 to 8);

[1] *Annales de l'Inst. Pasteur*, Dec., 1911, XXV, 12, p. 865.
[2] *Ann. de l'Inst. Pasteur*, Mar. 25, 1911, and Dec., 1911.

cresol, 1 per cent.; carbolic acid, 5 per cent.; or formalin, 10 per cent. The discharges should be received in a glass or earthenware vessel containing some of the germicidal solution. Then add more of the solution so that it shall be present in twice the volume of the excreta to be disinfected; let stand at least one hour, protected from flies. Masses are so difficult to penetrate that they should be broken up by stirring. It takes a carbolic acid solution some 12 hours to penetrate the interior of a fecal mass.

The sputum may be burned or boiled. Strong carbolic acid, tricresol, or formalin are also applicable.

The patient should have his own dishes, cups, spoons, glasses, etc., which should be scalded after each use. Remnants of lunch, especially meat, milk, gelatin, broths, and other organic food in which the infection may live and even grow should not be eaten by others. Such remnants may be burned or first boiled and then discarded. Those who nurse the sick should keep out of the kitchen on account of the risk of contaminating the food.

Towels, sheets, nightgowns, and all fabrics used about the patient should be disinfected either by boiling, or immersion for one hour in bichlorid of mercury, 1-1,000, carbolic acid, 2.5 per cent., or formalin, 10 per cent.

The water used to bathe the patient should be disinfected before it is allowed to run into the sewer. This may be done by adding sufficient carbolic acid or bleaching powder; the latter is cheapest and most practical.

Milk bottles must be kept out of the sick room. In any case, they should be scalded before returning to the dairy.

The thermometer should be kept in formalin or other suitable germicidal solution. Rectal tubes, especially in hospital practice, must be carefully disinfected each time before using.

The nurse must protect herself as well as others; a solution of bichlorid should be kept constantly at hand. Every time the patient is bathed, his mouth cleaned, or his buttocks washed, the hands must be disinfected and washed in soap and water. The nurse must exercise especial care if she is to go to the kitchen or to the ice-box, etc., as is frequently the case in private houses, where a special diet kitchen cannot be provided. The nurses, physicians, ward attendants, and others particularly exposed may protect themselves with preventive typhoid inoculations. The physician should be quite as careful as the nurse, not only so that he may not carry the infection to himself or other patients, but also that his practice may serve as a stimulating example.

At the conclusion of the case a general terminal disinfection of the room and its contents may be practiced. This is best done with formaldehyde gas, followed by a general mechanical cleansing.

Convalescents should not be given liberty until the danger of bacillus carrying has passed. This may be determined only by bacteriologic examinations of the stools and urine. Four successive negative results at intervals of several days are required before a conclusive report may be vouchsafed in the case of the stools. One examination of the urine is ordinarily sufficient.

The use of urotropin (hexamethylenamin) diminishes the incidence of bacilluria, and is becoming a routine practice.

Summary—Personal Prophylaxis.—The prevention of typhoid fever may be summed up in the word cleanliness—physical and biological cleanliness. By this is meant not only clean food, especially water and milk, but also cleanliness of person and environment. Typhoid fever has always prevailed where cleanliness is neglected and has diminished where it has been intelligently observed. It is true that typhoid bacilli do not breed in the rubbish and dirt of back yards and alleys, or in unkempt city lots, but these conditions in a city may be taken as an index of the general cleanliness of its inhabitants.

The eradication of typhoid fever is easier in cities than in country districts; clean cities now have less typhoid fever than the surrounding rural region. Cities can well afford extensive and expensive sanitary works which are beyond the financial possibilities of sparsely settled districts. If a clean water from natural sources is not available, then large volumes of a polluted water may be rendered reasonably safe for municipal use by slow sand filtration or by bleaching powder. Further, cities can afford to inspect their milk supply and to supervise the pasteurization of all that is not safe. These two measures would practically eliminate typhoid infection coming into cities in its food supply —especially if in addition to this a supervision is maintained over oysters and shellfish, and vegetables partaken in their raw state. Further, cities can well afford to employ skilled and experienced health officials and are financially able to engage the services of experts. On the other hand, each farmhouse represents, in miniature, all the problems with which the city deals by wholesale, and is often not financially able to meet its sanitary requirements. The country is the weakest link in our sanitary chain. Cities will find it a paying proposition to suppress flies, rats, and other vermin, which may be done much more easily than in rural or suburban conditions. This should be done not only on account of the suppression of typhoid fever, but other diseases thus conveyed. The city beautiful must also be the city clean in its cellars, garrets, back yards, empty lots, alleys, and stables.

To sum up, the main factors in the spread of typhoid fever in our large cities are: (1) water; (2) milk; (3) contact; (4) miscellaneous. In a city having a clean water supply the residual typhoid must be attacked along two definite lines, viz., improvement of the milk supply

and its pasteurization, and a warfare against the disease in the light of an infection spread from man to man.

The health officer should establish a laboratory for the early diagnosis of cases and for the discovery of carriers. The health officer should at once send a trained agent to every house from which a case of typhoid fever is reported. The visit should be made as early as practicable and with the object of seeing that the stools and urine are properly disinfected, patients isolated, milk bottles scalded, sick rooms screened, house placarded, visiting discouraged, and other necessary measures taken to prevent the spread of the infection. Convalescents should not be released until the absence of typhoid bacilli from the urine and stools has been demonstrated by four successive examinations. Carriers need not be indefinitely quarantined, but should be prohibited from engaging in any employment having to do with foods, or in which close personal contact, as in nursing, is required. Carriers should be instructed concerning the danger and educated to thoroughly wash and disinfect their hands, especially after a visit to the toilet. The health officer alone cannot eliminate typhoid fever from a city. He needs the help of the community. Much can be done through education. A stimulating leader may accomplish a world of good through voluntary effort, but in the end it requires comprehensive laws and an energetic enforcement of them, without fear or favor.

The personal prevention of typhoid fever resolves itself into boiling the water, if suspicious; partaking only of milk or fresh milk products that have first been pasteurized, and otherwise assuring oneself that all food has been thoroughly cooked. In addition to this, direct and indirect contact with persons who have the disease, or who are known to be carriers, must be avoided. Sanitary habits should be encouraged, especially the one simple precaution of washing the hands before eating, and of keeping the fingers and other unnecessary objects away from the mouth and nose. In certain circumstances in which there is unusual exposure protection may be had by increasing immunity through typhoid inoculations.

CHOLERA

The prevention of cholera corresponds to the prevention of typhoid fever. In the case of cholera vigorous measures have been rewarded with signal success. It is quite possible to live in the midst of a raging cholera epidemic without contracting the disease. Within recent years epidemics have been suppressed and the spread of the infection limited.

The home of true cholera is the delta of the Ganges, hence it is usually called "Asiatic cholera" to distinguish it from *Cholera nostras* or *Cholera morbus*. During the sixteenth, seventeenth, and eighteenth

centuries cholera was epidemic at various times in India. It is only in the nineteenth century that cholera has spread along the routes of trade and travel to Europe (first in 1830), Africa, and America in 1832. There have been four pandemics; one from 1817-1823, another 1826-1837, a third 1846-1862, and a fourth from 1864-1875. In 1832 it entered the United States by way of New York and Quebec and reached as far west as the military posts of the upper Mississippi. The disease recurred in 1835 and 1836. In 1848 it entered the country through New Orleans and spread widely up the Mississippi and was dragged across the continent by the searchers for gold all the way to California (1849). It again prevailed widely through this country in 1854, having been introduced by immigrant ships into New York. In 1866 and 1867 there were less extensive epidemics. In 1873 it again appeared in the United States, but did not prevail widely. In 1892 the great epidemic of Hamburg occurred, and the disease threatened to become pandemic in Asia, Africa, and Europe. Cases were brought by transatlantic liners to New York, and a few cases occurred in the city, but its spread was prevented by aggressive measures. Cholera has prevailed for years in the Philippines, but is now under control. While the home of cholera is in the tropics, there is scarcely a country in the world that has not been visited some time or other by the ravages of this fatal disease.

The incubation period of cholera is short, frequently 1 or 2 days, rarely over 5. The period of detention in quarantine is 5 days. One attack produces a mild grade of immunity which is not lasting. The disease is peculiar to man.

The Cause and Contributing Causes of Cholera.—The *Vibrio choleræ* or the "comma bacillus" of Koch is the undisputed cause of the disease. The conditions of infection, however, are complex. Not everyone who takes the specific microorganism by the mouth necessarily gets the disease, but without it there can be no cholera. Many cholera vibrios probably die in the acid juices of the stomach. There is, therefore, perhaps less danger in taking small amounts of infection during active digestion than upon an empty stomach, for it has been shown experimentally that cold drinks do not stay long in an empty stomach, but pass quickly through the pylorus. After the cholera vibrio has passed the pylorus and reaches the alkaline juices of the intestines, it may find ideal conditions for growth or may still have a hard struggle for existence. Here symbiosis must play a dominant rôle. It is well known in all cholera epidemics that a deranged digestion is an important predisposing factor to the disease. In the Hamburg epidemic a marked access of cases on Monday following the Sunday dissipations was noted. Raw fruits, crude fibrous vegetables, and other fermentable food, difficult of digestion, seem to favor the growth and multiplication of the cholera

vibrio in the intestinal tract. In the light of this view raw fruits and vegetables may often be the predisposing factor rather than the medium which conveys the infection. Just what the factors are that favor or handicap the growth of the cholera vibrio in the intestinal tract are undetermined. Pettenkofer stoutly maintained that the "comma bacillus" was only one of the factors in the etiology of the disease. He placed special importance upon the condition of the host and his environment, and considered at least three fundamental factors in his X, Y, Z theory. X is the germ, Y the host or soil, Z the environment. In this connection disease may aptly be compared to fermentation, in which X represents the yeast, Y the carbohydrate, and Z the temperature, moisture, reaction, and other essential conditions for the growth and activity of the yeast. Pettenkofer maintained that X without Y and Z would not produce cholera, that is, while the cholera vibrio was pathogenic in India or Hamburg (1892), where Y and Z were favorable, it would be harmless in Munich, where Y and Z were unfavorable. To prove this theory, he and his assistant, Emmerich, drank pure cultures of cholera after first rendering the stomach contents alkaline. Pettenkofer, then an old man, had a diarrhea; Emmerich, on the other hand, had a sharp attack from which he almost lost his life. Similar convincing experiments have occurred among laboratory workers, who have accidentally gotten pure cultures of cholera into their mouths. On the other hand, a number of persons who imitated Pettenkofer's experiment were not affected. Pettenkofer did not regard his own case as cholera, and insisted that the negative results lent confirmation to his theory of the importance of contributing factors (Y and Z).

Diagnosis.—The diagnosis of cholera depends upon isolation and identification of the cholera vibrio in pure culture. This has become comparatively simple, but great care must be taken not to confuse the true vibrio of cholera with a great host of other microorganisms which closely resemble it.

A presumptive diagnosis of cholera may be made by finding large numbers of comma-shaped bacilli in direct microscopic examination of stained preparations, or in hanging drops of the mucous flakes ordinarily found in cholera stools. This test is only presumptive, the final criterion being the biological reactions of the microorganism obtained in pure culture. The two reactions which are specific and reliable are Pfeiffer's phenomenon and agglutination.

Dependence should not be placed upon morphological characters, cultural peculiarities, or pathogenicity upon laboratory animals, for these do not furnish the means of certainly defining the cholera vibrio. For the isolation of the cholera vibrio agar is preferable to gelatin, formerly so much used. The suspected material should be planted upon

the surface of ordinary alkaline agar or upon Dieudonné's medium, using one of the small rice-like flakes or an equivalent quantity of feces.

Dieudonné's medium is prepared as follows:

Sol. A.—Equal parts of a normal solution of potassium hydroxid and defibrinated ox-blood are mixed and sterilized in the autoclave.

Sol. B.—Ordinary nutrient agar, exactly neutral to litmus.

Seven parts of B are mixed with 3 parts of A and poured into Petri dishes. The plates should not be used immediately after their preparation. Dieudonné recommends keeping them several hours in the incubator at 37° C., uncovered and face down, or to heat them for 5 minutes at 65° C. Equally good results can be obtained by keeping them 48 hours at room temperature. The surface of the agar should be slightly dry. Once in condition, the plates should be used in a period not exceeding 5 or 6 days.

Upon this medium cholera vibrios grow abundantly. On the contrary, the organisms which most often accompany them on plate cultures, especially *B. coli,* grow either very poorly or not at all.

When it is suspected that the cholera vibrios are few in number, they may be enriched by first planting in Dunham's solution. Approximately 1 c. c. of fecal matter should be placed in 50 c. c. of the peptone solution. This is incubated at 37° C., and in from 6 to 8 hours a loopful is taken from the surface and transferred to ordinary agar or Dieudonné's medium. Suspicious colonies are fished and studied further. A quick method of detecting carriers is given on page 108.

Kolle and Gotchlich have shown from a large number of observations that with strongly agglutinative serum, the power of which reaches 1-4,000, the agglutinative power for common vibrios, not cholera, does not, as a general rule, exceed 1-50 and rarely reaches 1-200; agglutination in dilutions of 1-500 has been only very exceptionally observed. On the contrary, the true cholera vibrios agglutinate in dilutions varying from 1-1,000 and 1-20,000. Therefore, with a specific agglutinating serum having a titer of 1-4,000, any organism which is agglutinated in 1-1,000 may be considered true cholera. Organisms agglutinating in dilutions of 1-500 and 1-1,000 should be regarded as doubtful.

In any critical case Pfeiffer's reaction (see page 389) should be tried. This is specific.

Modes of Transmission.—Cholera is spread by man from place to place. It follows the lines of trade and travel. Seaports are invariably first attacked. The epidemic at Hamburg in 1892 was brought to that port by immigrants on board vessels from Russia. There are many similar instances. In 1849 many a gold hunter found another Eldorado than the one he was searching for, as cholera was dragged

across the continent by the caravans seeking fortunes in California. The same thing takes place in the Indian pilgrimages to Mecca.

The cholera vibrio enters the digestive tract through the mouth. It is taken in the food and drink. Infected water is a frequent medium of transference, and probably the sole vector of the great epidemic outbursts. Cholera, however, may be transferred from man to man directly, also indirectly by flies, fingers, food, and all the innumerable channels from the anus of one man to the mouth of another that have been described in the case of typhoid.

In endemic or residual cholera, water-borne infection plays a minor rôle. This was well proven in the recent sanitary campaign against the disease in the Philippine Islands, in which the water was practically ignored and the disease conquered in the light of an infection communicated rather directly from man to man. Cholera was spreading rapidly despite active measures. Its progress was stopped by throwing a sanitary corps across a narrow neck of land some miles in advance of the march of the disease. Here a quarantine was established and persons held 5 days under observation before they were permitted to pass. The usual disinfection and other measures were practiced and the disease effectively stopped.

The cholera vibrio leaves the body in enormous numbers in the dejecta, also sometimes in the matter vomited. The cholera vibrio does not invade the blood and tissues generally, and, therefore, is not voided in the urine. Disinfection in this disease must, therefore, be concentrated upon the discharges from the bowels and mouths, at the bedside.

Water.—The cholera vibrio may live and even multiply in water. Koch in his original investigations found the organism in the foul water of a tank in India which was used by the natives for drinking purposes. It has been shown by experiment that the cholera vibrio may multiply to some extent in sterilized river water or well water; and that it preserves its vitality in such water for several weeks or even months. In recent times cholera organisms have been found not infrequently in the water of wells, water mains, rivers, harbors, canals, and even sea water (the North Sea near the mouth of the Elbe), which have become contaminated with the discharges of cholera patients. It is plain from the nature of the case that infected water must play a very large rôle in spreading this infection.

The Broad Street Case in London.—The earliest and now classic instance in favor of the water-borne theory we owe to the late Dr. John Snow. This is the well known Broad Street pump outbreak in London in 1854, an account of which will be found on page 815.

The best example of water-borne cholera is the Hamburg epidemic of 1892, which I was fortunate enough to see in part. In this case no link in the chain of evidence is missing. Cholera was brought to

Hamburg by immigrants either from Russia or France. The water of the Elbe was infected with their discharges. The *Vibrio choleræ* was readily isolated from the river water which was distributed throughout the city for drinking purposes without purification. The sewers of Hamburg emptied into the river Elbe near the water intake, which produced an increased concentration of the infection. An account of the epidemic will be found on page 819.

Other Modes of Transference.—The fact that water-borne infection is practically the only cause of the large cholera epidemics must not overshadow the importance of other modes of transmission. In addition to the violent outbreaks, cholera occurs in nests or smoulders like endemic typhoid. It is difficult to trace the connection between cases in endemic areas. Thus, a careful study of the cholera situation in Manila disclosed the fact that isolated cases would crop up at widely different points without any evident connection between them. Cholera carriers were suspected but not proved in this instance. At irregular intervals of several years the disease would gather force, and cases multiply, until it assumed epidemic proportions, it is believed entirely independent of the water supply. The way cholera was dragged across our continent by the "forty-niners," and its occurrence among the Mecca pilgrims, are instances of its spread largely independent of infected water.

Contact Infection.—Contact infection in cholera must not be underestimated. Persons frequently become infected by handling the dejecta or through freshly infected fomites, such as soiled linen. Direct transmission from person to person is not infrequent among physicians and nurses. In congested quarters, where many persons live under uncleanly conditions, contact infection plays an important part. The same thing may be seen on board vessels, in which case the disease may be confined to the firemen, stewards, or some other limited group who are required to live in close contact with each other. Epidemic outbreaks due to contact infection have been recorded, such as the 30 cases which occurred in the fall of 1892 in Boizenburg.

Cholera is not highly "contagious," for physicians, nurses, and others in close contact with patients need not become infected provided intelligent measures are adopted. On the other hand, there is great danger of the spread of the disease through devious and hidden routes, as is the case with typhoid and dysentery. Washerwomen and those who are brought in very close contact with the linen of cholera patients or with their stools are prone to contract the disease. Koch, in his original investigations, found that the "comma bacillus" may multiply rapidly upon the surface of moist linen.

Milk may be contaminated, but is probably not a frequent medium of infection, for the reason that its acid reaction is inimical to the

cholera vibrio. Green vegetables and fruit that have been washed in an infected water may convey the disease. The bacilli live on fresh bread, butter, and meat for from 6 to 8 days.

Flies, Etc.—It has been shown that the cholera vibrios may live in the intestines of flies for at least 3 days, and these and other insects may also spread the infection mechanically. The cholera vibrio is a frail organism and dies rapidly when dried or exposed to light and other injurious influences. Infection through the air is, therefore, not to be dreaded. Fomites, such as bed and body linen or other objects, including floors, walls, toys, etc., contaminated with the dejecta, can be regarded as possible sources of infection. There is, however, a special limitation in this case, owing to the fact that this organism is so readily destroyed by desiccation and crowded out by saprophytic microorganisms. Thus, as a rule, only fresh dejecta and freshly contaminated objects are liable to convey the infection.

Bacillus Carriers.—The cholera vibrios are passed in the feces during the early part of the disease. They usually disappear after the fourth to the fourteenth day, but may remain a much longer time. The following are the longest cited by Pfeiffer:[1]

PERSISTENCE OF CHOLERA VIBRIOS IN STOOLS OF CONVALESCENTS, OR BACILLUS CARRIERS

Name of Observer.	Longest Duration (days).	Name of Observer.	Longest Duration (days).
Guttman	10	Kolle	48
Lazarus and Pulicke	12	Donitz	49
Michailow	12	Abel and Clausen	15
Simonds	18	Pfeiffer	13
Rumpel	24	Bürgers	69
Rommelaere	47		

McLoughlin found bacillus carriers numerous in epidemic centers. Thus he found 6 to 7 per cent. of carriers among healthy individuals living in the infected neighborhoods in Manila. On the other hand, carriers were exceedingly rare in neighborhoods having few cases. Persons in good health may harbor the cholera organism in their intestines. Cholera carriers, therefore, play a similar rôle to typhoid carriers in spreading the infection. Less, however, is known concerning cholera carriers than typhoid carriers.

Several different methods for the detection of cholera carriers are applicable. All of them are based upon the facility with which the vibrio grows upon Dunham's solution. Particles of feces are planted in this medium and subsequently examined for comma-shaped microorganisms. If found, the diagnosis is presumptive. Pure cul-

[1] Hygienische Rundschau, February, 1910, Vol. XX, No. 4.

tures should then be made and studied for agglutination. See page 104.

The routine bacteriological examination of immigrants from cholera-infected ports, as practised at the Quarantine Station at New York, in 1912, was as follows:[1]

1. Inoculation of feces into Dunham's peptone solution (at 37° C.).

2. Subinoculation at the end of six hours of one loop of the surface growth into a second Dunham's peptone tube.

3. Examination of a smear taken from the surface growth of the second Dunham's peptone tube, after it has been incubated six to nine hours at 37° C.

Bendick uses a modified Dunham's solution containing sodium carbonate, 1 gram; saccharose, 5 grams; and phenolphthalein solution, 5 c. c., in addition to the usual amount of water, peptone and salt. The cholera vibrios ferment the saccharose; the acid produced unites with the sodium carbonate and the medium becomes neutral, hence the red color of the phenolphthalein disappears.

Immunity and Prophylactic Inoculations.—The immunity produced by an attack of the disease is of short duration. Attempts have been made to produce an artificial immunity by the injection of cholera cultures. These were first made by Ferran of Spain in 1884, but the cultures used by him obtained directly from cholera stools were not pure. Haffkine tested the method on a large scale in India; over 40,000 persons were inoculated with attenuated cultures up to 1895. Haffkine proceeded in accordance with the well known methods of Pasteur in anthrax, by using two vaccines of different strengths. The first was obtained by growing the culture at a heightened temperature, which produced a very attenuated strain. The second contained living vibrios weakened by passage through guinea-pigs. The reactions produced were generally slight in degree and consisted of a brief elevation in temperature, headache, malaise, as well as redness, swelling, and pain at the site of injection. The results were not clear cut on account of the difficulty of comparing the disease in the inoculated with suitable controls. However, the general impression is that the method has some prophylactic value. This opinion has been confirmed by the later work in various parts of India, where, up to the year 1899, of 5,778 inoculated persons, only 27 had cholera and 14 died, whereas, of 5,549 non-inoculated, 198 had cholera, of which 124 died. Kolle showed that the blood serum of the inoculated persons contains a specific bacteriolysin similar to that contained in the blood serum of those who have recovered from the disease. Kolle uses 2 mg. of an agar culture suspended in 1 c. c. of physiological salt solution and killed at 58° C. for one hour for the first injection, and twice this

[1] Bendick: *Jour. of Am. Pub. Health Assn.*, I, No. 12, 906, Dec., 1911.

dose (4 mg.) for the second; 0.5 per cent. of phenol is added as a preservative. The immunity produced by these protective inoculations lasts a long time, but after a year the specific antibodies begin to diminish in the blood serum.

There seems to be little doubt in Japan concerning the value of the protection afforded by the inoculation of dead cultures, for in the district of Hiogo, during the epidemic of 1902, 77,907 persons were inoculated. Of these 47, or 0.06 per cent., took cholera, and 20, or 0.02 per cent., died, whereas, among 825,287 persons not inoculated, 1,152, or 0.13 per cent., took the disease, and 863, or 0.1 per cent., died. It is especially noteworthy that all the cases among the inoculated group were in those who received an injection of 2 mg. of the dead culture. Later 4 mg. were used, and in this group no cases occurred.

Protective inoculations as a prophylactic measure against cholera will never be popular or necessary in communities with sufficient sanitation. It may, however, be of value in camps, armies on the march, for physicians, nurses, ward tenders, and others especially exposed.

Quarantine.—Cholera is an infection which fully justifies maritime quarantine practice. The disease may be blocked by a careful system of inspection, detention, and disinfection at the seaport. In order for maritime quarantine to be effective for cholera, it must have the assistance of a bacteriological laboratory to diagnose cases and recognize carriers. A strict watch must be kept for mild and ambulant cases of the disease.

In the summer of 1912 the quarantine authorities at the large seaports on our Atlantic littoral examined about 34,000 specimens of bowel discharges from passengers and crew from cholera-infected ports. At the New York quarantine the cholera vibrio was isolated from 28 persons sick with the disease, and 27 healthy persons were found to be discharging vibrios in their feces. These carriers could not have been discovered except by laboratory examination. Seven cases of cholera were detected at other ports by the same methods. There can be no doubt that the adoption of this measure kept cholera out of the country.

The Foreign Inspection maintained by the United States Government during the epidemic of 1892-93 was a convincing demonstration of the value of this service as one of the safeguards against cholera. Officers of the Public Health and Marine Hospital Service stationed at foreign ports supervised the water and food supply of the departing vessels, inspected the crew and passengers as to their health; those coming from infected localities were detained under observation 5 days before they were permitted to embark. On practically none of the vessels complying with these requirements did cholera appear,

whereas it broke out comparatively frequently on vessels which did not comply with the restrictions, but sailed from the same ports under similar conditions. A similar experience demonstrating the value of a sanitary inspection of vessels leaving an infected port was demonstrated in the Philippines, where, since the American occupation, cholera has been kept off the returning transports and its interisland spread has been checked by a sanitary supervision of vessels at both the ports of departure and arrival.

Personal prophylaxis requires, first of all, scrupulous cleanliness on the part of the person and his surroundings. Those who handle cholera patients, their dejecta, or infected articles must carefully disinfect their hands each time, and should under no circumstances eat or drink anything in the sick room. During cholera times all water and food of every description should be boiled or thoroughly cooked just before it is partaken of. Great care must be exercised that the water or food does not become infected after it has been boiled or cooked. The usual measures should be taken to guard against flies and other vermin. With strict attention to these measures, it is possible to avoid the infection. In addition, however, attention to general hygiene and especially to the character of the food and regularity of meals should be given. Slight attacks of indigestion and diarrhea should receive prompt medical attention.

Summary—Prevention.—Preventive measures should first of all be focused upon the cholera cases in order to prevent the spread of the infection at the bedside. This includes early and controlled diagnosis.

Cholera patients should be cared for in special hospitals where all these necessary measures may be carried out by trained assistants. The infection in cholera stools may be destroyed with formalin (10 per cent.), carbolic acid (5 per cent.), milk of lime (1 to 8), or chlorinated lime (3 per cent.).

Persons leaving a cholera region should either be detained in quarantine for 5 days or be watched this length of time after arrival at the place of destination. This may be accomplished by requiring them to report twice daily to the sanitary authorities. It is unnecessary to disinfect merchandise shipped from a cholera town.

For the control of a cholera outbreak it is important to require that all cases, as well as all suspicious cases, be reported. A bacteriological laboratory is necessary to confirm the diagnosis and arrangements must at once be made to isolate the cases and to disinfect the dejecta, the body and bed linen, and other materials. Convalescents are not released until two successive examinations at 5-day intervals show the absence of the cholera vibrios.

On account of the frail character of the vibrio a general disinfection of the house is not necessary in cholera. The room itself may

be treated with formaldehyde or the surfaces washed down with a bichlorid solution or one of the alkaline coal-tar creosotes. The water-closets may be disinfected with formalin, carbolic acid, milk of lime, or chlorinated lime. Spoons, cups, saucers, and remnants of food should be treated as in the case of typhoid. Otherwise the prevention of cholera is a strict counterpart of that of typhoid.

A summary of the preventive measures necessary to control an outbreak of cholera are: centralization of authority in one person; establishment of a system of securing and reporting information; organization of the personnel for the sanitary work; enactment of necessary ordinances; house to house inspection; safe disposal of feces of entire population; provision for a safe water supply; supervisory control of food and drink; a search for, and control of carriers; isolation and care of patients in special hospitals; separate hospitals or wards for suspects; a laboratory; detention camps or barracks for those desiring to leave the infected area; disinfection, etc. For further discussion concerning the control of epidemics, see page 319.

DYSENTERY

Classification.—For the purpose of prevention we may consider all dysenteries under three heads: (1) bacillary dysentery, (2) amebic dysentery, (3) symptomatic dysentery.

Bacillary dysentery is an acute infectious disease caused by the *B. dysenteriæ*, an organism that closely resembles the typhoid bacillus in cultural respects. It differs from typhoid in that it has limited or no motility. More fundamental differences are found in its biological properties, such as specific agglutination and pathogenic power. There are at least two well recognized types of *B. dysenteriæ*. One corresponds to the original organism discovered by Shiga in 1897 in the Japanese epidemic, and the other to that found by Flexner in Manila. The Shiga bacillus does not ferment mannite, while the Flexner ferments that "sugar" with the production of acid. Further, the two organisms differ in their properties of agglutination toward specific sera. A very strong endotoxin may be extracted from the Shiga type which, when injected intravenously into rabbits, produces a fatal intoxication with a faithful reproduction of the symptoms and lesions of bacillary dysentery. Kraus and Dörr and also Todd have found that the Shiga strain produces such a soluble toxin, which is not the case with the Flexner strain.

Amebic dysentery results from infection with the *Entamœba hystolytica*. There are marked differences between the amebic and the bacillary types of the disease. The former is a chronic infection which starts insidiously, is characterized by relapses and recurrences, is fre-

quently associated with sequelæ, such as liver abscesses, and occurs sporadically or in endemic foci, mainly in the tropics. Epidemic outbreaks of the amebic form of dysentery are not known. Bacillary dysentery, on the other hand, is an acute febrile disease, usually self-limited, with marked symptoms of toxemia, sudden onset, no sequelæ, and occurs in widespread and severe epidemics. The bacillary disease occurs in the temperate regions as well as in the tropics, and is almost always the cause of dysentery outbreaks in ships, camps, jails, etc. The lesions of the two diseases also differ markedly. In amebic dysentery the ulcers are undermined, whereas in the bacillary disease the inflammation is diffuse and of varying grades of severity, which may reach coagulation necrosis or gangrene. There are also notable differences in the treatment; for example, ipecac given early or rectal injections are of service in amebic dysentery, but are of questionable use and may even do harm in the bacillary form. So far as prevention is concerned, however, both diseases may be regarded as intestinal infections entering by the mouth, and therefore the prophylaxis is practically the same and corresponds closely to that of typhoid or cholera.

Under symptomatic dysentery are grouped all other conditions with dysenteric symptoms resulting from a great variety of causes.

Modes of Transmission.—The dysentery bacillus enters the body by the mouth and leaves the body in the alvine discharges. So far as known, the dysentery bacillus does not penetrate deeply into the tissues, and is seldom found in the circulating blood. It therefore does not appear in the urine.

The infection is transferred from man to man directly or indirectly in precisely the same ways described for typhoid. Undoubtedly drinking water frequently contains the infection, and well marked water-borne epidemics have been reported in recent years, particularly in Japan. Contacts, food, and flies also play an important rôle. The epidemiology of bacillary dysentery is about the same as that of typhoid. It is largely a summer disease. In wars it used to cause great ravages; as in the Crimean war, our own civil war, the Franco-Prussian war, and the recent Russian-Japanese war. Overcrowding, lack of cleanliness, and other unhygienic conditions favor the spread of bacillary dysentery, so that it is sometimes called famine, asylum, ship, or jail dysentery. The mortality varies greatly, from 6 or 7 to 26 or 30 per cent. Bacillus carrying in dysentery occurs, and probably plays a more important part in spreading the disease than we now suspect. As a rule, the bacilli soon disappear from the stools in the light cases, but Shiga has found them more persistent in some instances. von Drigalski reports an outbreak in Germany caused by a returning soldier. Recent convalescents are particularly apt to spread the infection.

The *Entamœba hystolytica* is also taken in by the mouth and

passed by the bowels. It probably exists in its free living state in water, upon vegetables and fruits, and other moist surfaces. There is some suspicion that the buds of the entameba may be carried by the air. There are still large lapses in our knowledge concerning the free living stages, and other facts in the life history of the ameba, so that our preventive measures lack finality.

Resistance.—The dysentery bacillus has about the same resistance to germicides and other unfavorable conditions as the general class of spore-free bacteria. It dies in about 8 to 10 days when dried. It may live for months when moist. It is sensitive to acids. Phenol, 0.5 per cent., kills the dysentery bacillus in 6 hours, 1 per cent. in 30 minutes, 3 per cent. in 1 to 2 minutes. Bichlorid of mercury, 1-1,000, kills it at once, and direct sunlight in about one-half an hour. I have found certain strains of the dysentery bacilli somewhat more resistant to heat than the typhoid bacillus. They are killed with certainty at 58° C. for one hour, or at 60° C. for 20 minutes. The dysentery bacillus resists cold and may live for months when frozen.

The *Entamœba hystolytica* is probably less resistant to heat and germicides than the *B. dysenteriæ*. Our knowledge concerning the effects of drying, sunlight, and other deleterious influences is still uncertain.

Immunity.—The susceptibility to dysentery varies greatly. This is doubtless due in part to the bacterial flora of the intestinal tract as well as the conditions of the intestinal mucosa. Symbiosis must play a very important rôle either in permitting or hindering the dysentery bacillus to grow in the intestinal tract. There is still a question whether a true immunity is acquired by one attack of bacillary dysentery. This seems probable, although it is not unusual for a person to have two or more attacks of dysentery in one season. Kolle looks upon this as an exacerbation of a chronic type brought on by errors of diet, exposure, etc. The experiments on animals indicate that dysentery probably belongs to that group of diseases which leave a certain amount of protection after one attack. A definite and high grade of immunity can be produced experimentally in several of the lower animals. Upon this question, however, we need light. Horses may be immunized to a high degree, and their sera contain a certain amount of antitoxin and other antibodies. This serum has been used in treatment, but has no particular value as a preventive. There is evidently no immunity in amebic dysentery.

Personal Prophylaxis.—To avoid dysentery the two essentials are: scrupulous cleanliness and the boiling of all water and cooking of all food that passes the mouth. The usual precautions against flies and vermin, and care as to personal hygiene, especially diet, are indicated.

Bacillary dysentery is a common disease in infants, and it would be

a wise precaution to consider all cases of infantile diarrhea as infectious and to take precautions accordingly.

Dysentery should be included in the notifiable diseases and laboratory aid furnished by the Board of Health to assist diagnosis. Cases should be isolated in the same sense that cases of typhoid are isolated and disinfection practiced at the bedside. Outbreaks in institutions should always be investigated and vigorous measures taken to check further spread and to prevent recurrences. In all respects the prevention of dysentery is a close parallel to that of typhoid.

HOOKWORM DISEASE

(*Uncinariasis or Anchylostomiasis*)

Theoretically the prevention of hookworm disease is comparatively simple, for here we have an infection of which we know the parasite and its life history, its mode of exit and entrance into the body, and we possess a satisfactory cure for the disease within reach of all. Practically, however, we have ignorance, apathy, poverty, and uncleanliness to deal with before satisfactory prevention, much less eradication, can be achieved. It is now plain that hookworm disease presents a sanitary problem of first magnitude, not alone in our southland, but in practically all tropical and subtropical countries. Further, there is a large economic and industrial aspect to this question in medical biology.

Distribution.—Hookworm disease encircles the globe in the tropical and subtropical climes; it diminishes toward the temperate regions. It is not endemic in the colder latitudes, except in mines, especially those of Wales, Germany, Netherlands, Belgium, France, and Spain. The infection belts the earth in a zone about 66° wide, extending from parallel 36 north to parallel 30 south latitude. The amount of infection is great in American Samoa, where it is found in 70 per cent. of the population; in the southern two-thirds of China, in 75 per cent. of the population; in India from 60 to 80 per cent. of the 300,000,000 population have the disease; in Ceylon, 90 per cent. in many parts; in Natal, 50 per cent. of the coolies on sugar and tea estates; in Egypt, 50 per cent. of the laboring class; in Dutch Guiana, 90 per cent. in many parts; in British Guiana, 50 per cent. of all; in Colombia, 90 per cent. of those living between sea-level and 3,000 feet, which includes most of the population; in 1904 the Porto Rican Anemia Commission found that 90 per cent. of the rural population of Porto Rico were infected. Stiles estimates that in this country 2,000,000 individuals have the infection from the Potomac to the Mississippi, along the Atlantic littoral and the Gulf states. In some German mines from

30 to 80 per cent. of the miners have been found to be infected. Gunn[1] has shown that from 50 to 80 per cent. of those working in the California mines are infected. It is probable that all the older mines employing foreign laborers sooner or later become endemic foci.

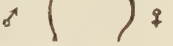

Fig. 14.—Hookworms, Natural Size.

In 1879 an outbreak of hookworm disease (miner's anemia) occurred among the laborers in St. Gothard's tunnel. This aroused the interest of the whole scientific world. The polluted soil of the tunnel was found to be impregnated with the eggs and larvæ. Interest in the disease in this country was aroused through the work and enthusiasm of Stiles.

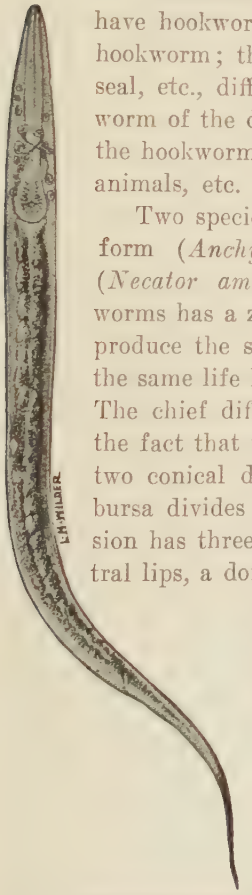

Fig. 15.—Hookworm Embryo.

Varieties of Hookworm.—Almost all mammalian animals have hookworms, but each host species has a different kind of hookworm; that is, the hookworms of the dog, fox, horse, the seal, etc., differ from each other, and are specific. The hookworm of the dog will not infest man or other mammalian host; the hookworms of man do not develop to maturity in the lower animals, etc.

Two species of hookworm are found in man—the old world form (*Anchylostoma duodenale*), and the new world form (*Necator americanus*). The distinction between these two worms has a zoölogical rather than a practical bearing, for both produce the same symptoms, require the same treatment, have the same life history, and call for the same preventive measures. The chief differences between these two hookworms consist in the fact that the old world form has one pair of ventral hooks, two conical dorsal teeth, and the posterior ray of the caudal bursa divides two-thirds its way from the base, and each division has three tips (tripartite). The new world form has ventral lips, a dorsal median tooth, and one pair of dorsal and one pair of ventral lancets deep in the buccal capsule. The posterior ray of the caudal bursa divides at its base and each division has two tips (bipartite).

According to Stiles, the vast majority of cases of hookworm disease in man in the United States are due to the new world form (*Necator americanus*).

Modes of Transmission.—The usual mode of transmission, perhaps in 90 per cent. of the cases, is through the skin. The infection may also be taken by the mouth in drinking water or soiled food, or from contaminated objects, such as dirty fingers. It has been

[1] *Jour. A. M. A.*, Vol. LVI, No. 4, Jan. 28, 1911, p. 259.

shown by experiment that animals can be infected by drinking water containing the embryos. While this source of infection plays a minor rôle, it is not to be disregarded.

The infection leaves the body exclusively in the feces, which contain the eggs of the parasite.

The Parasite.—For a correct understanding of the prevention of hookworm disease it is necessary to have a knowledge of the essential features of the life history of the parasite.

Hookworms are round worms (nematodes) belonging to the subfamily *Uncinariinæ*. The adult worm is about one-half to three-quarters of an inch long, and about the diameter of a wire hairpin.

The adult hookworm lives in the intestinal tract, usually in the small intestine. It attaches itself to the intestinal wall, wounds the mucosa, sucks blood, eats the epithelium, and probably produces a toxic substance which injures the host.

The female worm lays a prodigious number of eggs in a never-ending stream, which pass from the host in the feces. The embryo does not mature within the egg except in the presence of oxygen. Hookworm embryos, therefore, do not undergo full development until the eggs are discharged into the outer world. On the other hand, the eggs of *Strongyloides stercoralis,* the parasite of Cochin-China diarrhea, contain fully developed embryos in the freshly passed feces. The hookworm embryos become mature within the egg in 6 to 8 hours in the presence of moisture, warmth, and oxygen. It is, therefore, necessary to examine the fresh stools in order that this difference between the two infections may be of value in differential diagnosis.

Under favorable conditions the embryo escapes from the egg and becomes a larva in about 24 hours. This free-living larva exists and moves in moist soil and feeds upon the organic matter found there. In the course of two days or more the larva sheds its skin (ecdysis) and thus passes to the first molt. The larva continues as a free-living parasite, and in about a week again sheds its skin, but now continues to live encysted inside this discarded skin. This is the second ecdysis and this encysted larva no longer takes food. This stage in the life history of the parasite is of special importance for the reason that it is capable of piercing the skin; that is, it is the infecting stage. In this condition the parasite may live in a dormant condition for five months, perhaps longer.

The hookworm larva passes in all through five ecdyses or molts. Two of them occur during its free-living stage and three of them during its residence in the host. With each ecdysis the larva approaches more nearly the appearance and structure of the adult worm.

The larva has a slow motion and under favorable conditions probably travels a number of yards, increasing the radius of soil pollution.

The larva pierces the skin and passes by a circuitous route to the intestinal tract. The parasite may enter the skin at any place, but it usually goes through the soft and thin skin between the toes. In its passage through the skin the larva produces an inflammatory reaction (ground-itch) which results partly from the irritating action of the presence of the foreign body, but mainly from the bacteria carried along with the larva. These primary lesions may consist of a few itching papules or pustules to a severe dermatitis. Of 4,741 patients questioned by Ashford, King, and Gutierrez in Porto Rico, 4,664, or about 98 per cent., gave a history of ground-itch, which is now recognized as the first stage of the disease.

The fact that the infection with hookworm disease is usually contracted through the skin was discovered by Looss in Cairo, Egypt. He also unraveled the course of the parasite from the skin to the intestines. This brilliant discovery, which is one of the romances of medical biology, is the foundation upon which prevention against the infection depends. In 1895 Looss accidentally spilled a drop of water containing many larvæ upon his hand, and noted that they disappeared, leaving their delicate sheaths behind them. Seventy-one days subsequently he developed intestinal uncinariasis. The experiment was then repeated upon a volunteer, and hookworm eggs appeared in his stools in 74 days. Claude Smith found eggs in the feces 6½ weeks and 7 weeks after experimental skin infection on two persons with the American parasite (*Necator americanus*).

The wanderings of the parasite from the skin to the intestine were worked out by Looss partly by placing larvæ upon an amputated leg and also by studying the question upon puppies. The hookworm larva usually pierces the skin through a hair follicle, enters the subcutaneous tissue, and then finds its way through the lymphatics to the neighboring lymph nodes. The larvæ are able to squirm through the lymph nodes, pass to the thoracic duct, and thence to the vena cava and the right heart. From the right heart they are carried in the blood stream to the lungs. The larvæ are too large to pass the capillaries of the lungs. They pierce the capillary walls and appear in the alveoli and are now, to all intents and purposes, again in the outer world. They pass up the bronchi and trachea to the throat, whence they are swallowed, and finally lodge in the small intestines. During their travel through the body they pass through three ecdyses.

The adult worm attaches itself to the mucous membrane by means of the powerful buccal lancet. The epithelium is drawn into the buccal cavity as though by a powerful suction. The worms are usually found in the small intestine, especially in the jejunum, less often in the duodenum, and rarely in the ileum and lower reaches of the intestinal tract; they are occasionally met with in the stomach.

The parasites imbibe large amounts of blood, some of which passes through the worm unaltered. The wound continues to bleed after the worm releases its hold, owing perhaps to a hemolytic substance in the mouth parts of the parasite. The worm does not remain fastened to one place indefinitely, but releases its hold and attaches itself anew. This produces numerous minute wounds, favoring secondary infections. The hookworm probably produces a poison which is absorbed and which accounts, in part, for the anemia and other symptoms of the disease. The severity of the symptoms bears no definite relation to the number of worms. The number varies greatly in individual cases; from one or two to thousands. Sandwith counted 250 worms and 575 bites in one case; 2,000 are not an uncommon number. The Porto Rico Commission counted as many as 4,600 passed by one individual.

Immunity.—There is no acquired immunity to this disease. There is, however, a definite racial immunity, as shown by the negroes and the Filipinos, who are often infected but have comparatively slight symptoms. Stiles found that in this country the negro is the great reservoir for hookworm disease in that he is frequently infected but slightly affected. Perhaps the negro has had the disease so many generations in Africa that he has become immune. It is conjectured that the infection was brought to America through the negro slave trade. Hookworm disease lowers resistance and greatly increases the chances of other infections, especially tuberculosis. The secondary results are often more disastrous than the primary effects.

Resistance of the Parasite.—The adult worm in the intestinal tract may be benumbed or killed with thymol, betanaphthol, chloroform, gasoline, eucalyptus oil, and other of the usual vermifuges.

From the standpoint of prevention, it is more important to know the resistance of the eggs and larvæ during their free-living stages. Stiles and Gardner have shown that the soil under and around privies is not entirely free from infection with hookworm even five months after the privy was last used, although the infection is considerably reduced at the end of four months. When the fecal matter has undergone decomposition under water most of the hookworm eggs are dead in about ten weeks, though some still survive, but probably all are dead in three months. It would not be safe to use such material as a fertilizer in less than three months. The larvæ may live in water at least thirty days. The encysted stage is most resistant, surviving five months; perhaps longer.

The larvæ are readily killed by dryness and freezing. The infection was once considered to be dust-borne, but the fact that the parasites are killed by drying renders the danger from dust negligible. The fact that freezing kills the larvæ largely explains why the disease is not endemic in this country north of the Potomac.

It has been shown that chlorinated lime fails to kill hookworm eggs in 22 to 40 hours. Schüffler kept the larvæ alive almost four months in water with two or three drops of a one per cent. quinin solution to 10 c. c. Oliver found that sea water killed the larvæ in 37 minutes.

Prevention.—The prevention of hookworm disease consists in preventing pollution of the soil and in treating existing cases so as to diminish the amount of infection. The principles of prevention are easy in theory, but their application is difficult in practice on account of the widespread and enormous amount of the disease. The suppression of hookworm disease means the social and economic uplift of nations, the education of millions of people, and an entire change in their daily hygienic habits. Education of the masses is an important factor; calling for coöperation between the health authorities, civic forces, the medical profession, schools, and philanthropic agencies; it is something for the preacher and teacher.

Soil Pollution.—The prevention of soil pollution is the essential factor; it is the key to the situation. This one line of prevention would blot hookworm disease out of existence. This requires the building of proper privies, and insisting upon their being used in country districts. In warm countries direct pollution of the soil is much more common and also much more dangerous than in cold countries. Add to this the custom of going barefooted and we have all the factors necessary for the dissemination of hookworm infection.

Stiles estimates that 68 per cent. of the rural homes in the South are without privies. Even some schools do not have these accommodations, and are, therefore, hotbeds of infection. For the care and disposal of night soil see chapter on sewage.

The Eradication of the Infection in Man.—Hookworms may be expelled from the intestinal tract by the use of thymol, betanaphthol, or other anthelmintic. The eradication of the infection through the treatment of all infected persons is an essential factor in preventive measures. The usual treatment is as follows: Saturday evening a full dose of magnesium sulphate or other purge is given to permit direct access of the thymol to the worms, which are often imbedded in the mucus or chyme. The object is to treat the parasite and not the host. On Sunday morning, at 8 o'clock, 2 grams (30 grains) of thymol, for an adult, finely powdered in capsules, are given by the mouth. Two hours later, at 10 o'clock, 2 more grams are administered; and at 12 o'clock another dose of salts. During the treatment it is important to avoid alcohol, fats, and oils, as thymol is soluble in these substances and they are, therefore, dangerous, as they thus favor absorption. The treatment is repeated every Sunday until the eggs disappear. One treatment usually suffices. The Porto Rican Commission sometimes

found it necessary to use two, three, four, and up to eleven treatments.

The eradication of the infection in man was carried out on a wholesale scale by the Porto Rican Anemia Commission, consisting of Ashford, King, and Gutierrez. Their methods were highly successful and will doubtless serve an equally useful purpose in other places. They established a clinic for the microscopic diagnosis and free treatment of the disease. The good results of treatment spread rapidly, so that the facilities of the clinic were soon taxed to its utmost capacity. Not the least important function of the clinic was to educate the profession as well as the people. In a little while the clinic was moved to another point, and so on, until it gradually covered the entire island.

Education.—Education is one of the most important factors in eradicating hookworm disease, for the reason that its final control depends upon improvements in the sanitary habits of the people, especially in the rural districts. To change the daily habits of half a nation is an uplift that requires time and patience. It is perhaps best to begin with the school children; even then it will take a generation for results. Very little can be accomplished by force, and, if the customs and prejudices of the people are ignored, the reformer and benefactor meet with rebuff and failure. It is a good idea to have a public health day or a public health week in the schools, during which time lectures and educational work upon hookworm, typhoid, tuberculosis, and other prevalent infections are considered. The children carry the lesson into the home. Pamphlets, posters, lectures, exhibits, and popular articles in the magazines and newspapers all contribute their share. The medical profession in the infected areas may need instruction and a little prodding to awaken interest in the problem. In the popular education on health matters the medical profession should lead, especially through the health authorities. This has also become one of the manifest duties of the practitioner.

Cleanliness.—After all, the prevention of hookworm disease is a question of decency and cleanliness. Water sometimes carries the infection, hence it should be clean or cleansed by filtration or boiling. Soiled hands may carry the infection to the mouth, hence they should be washed before eating. Vegetables fertilized with night soil may be infected. This practice is not clean and should be forbidden, especially in the case of those vegetables usually eaten raw. With cleanly habits there would be no soil pollution, and the disease would be checked.

Personal Prophylaxis.—Personal prophylaxis consists in wearing shoes and otherwise avoiding contact with the infected soil. Brick makers, miners, and others compelled to work in infected soil should wear gloves. Other measures, such as boiling the water, eating only cooked or clean food, washing the hands, and avoiding the infected area,

have either been dwelt upon or are too obvious to need further emphasis.

Immigration.—An important factor in the spread of hookworm disease in the United States is immigration. Every country that brings laborers from infected regions is bringing in a constant stream of infection. California has established quarantine measures against Indian coolies, 90 per cent. of whom are infected.

Collateral Benefits.—The best part of a hookworm campaign is the collateral good it does. This applies as well to a sanitary campaign directed against almost any disease. The suppression of hookworm disease will diminish the amount of tuberculosis, typhoid fever, dysentery, and other infections. Thus, in Bilibid prison, Manila, the death rate was formerly excessive—234 per thousand when the Americans took charge. This was reduced to 75 per thousand by sanitary measures, such as boiled water, screens, disinfection, improved food, less crowding, better air, more sunlight, etc., but despite these sanitary improvements the death rate could not be hammered down below 75 per thousand. Then it was found that many of the prisoners were infected with hookworms. Thymol was administered and the death rate fell to 13.5 per thousand. Another instance of the collateral benefits resulting from sanitary work is the plague campaign in San Francisco, which cut typhoid fever in half, although no special attention whatever was paid to the latter disease. The purification of the water supply in Hamburg by filtration cut down the general death rate and diminished the morbidity of diseases not water-borne. One of the most encouraging phases of sanitary work directed against tuberculosis, typhoid fever, and hookworm disease is the assurance that a successful campaign will result in fundamental and permanent control or eradication of other communicable diseases. The prevention of tuberculosis deals especially with personal hygiene, and the prevention of typhoid fever and hookworm with the sanitation of the environment. The combination of the two, therefore, embraces almost the entire range of preventive medicine.

CHAPTER III

DISEASES SPREAD LARGELY THROUGH DISCHARGES FROM THE MOUTH AND NOSE

TUBERCULOSIS

Tuberculosis is the most frequent and widespread of all the major infections. In this country 9 per cent. of all deaths, and in Germany 12 per cent., are caused by tuberculosis. The toll falls heaviest during the period of life of greatest usefulness—thus 30 per cent. of all deaths between the years of 15 and 60 are due to pulmonary tuberculosis alone. Naegeli, from a careful examination of a large number of bodies in Zurich, found evidence of tuberculosis in over 90 per cent. The lowest figures based on the evidence of pathologic anatomy are those of Bitzke, who examined 1,100 bodies in Berlin. In children under 15 he found evidence in 27.3 per cent., and in persons over 15 58.2 per cent. The difference between Naegeli's figures and Bitzke's is due to a difference in the interpretation of the pulmonary scars and adhesions at the apices, and the small fibrous nodules in the lungs. Bitzke does not consider such lesions as of tuberculous origin, and leaves them out of his figures. If these were included, his percentage would also be very much higher. The frequency with which we become tuberculized is indicated by the fact that practically all persons more than a few years old give the von Pirquet cutaneous reaction.

In the United States it is estimated that 160,000 persons die each year of tuberculosis. Of the 90,000,000 people now living in this country, it is estimated that 8,000,000 are doomed to die of tuberculosis, unless the disease is checked. The loss in life and treasure is appalling. It is, therefore, most encouraging that preventive measures based upon modern conceptions of the disease as a communicable infection are giving encouraging results.

Tuberculosis began to decline before the nature of the infection was known. The decline is gradual. Modern methods have so far made little apparent impression upon the gross amount of the infection. The social and economic conditions of the

mass of the population must be improved before any great decline in the mortality rate can be expected, as will presently be pointed out.

Tuberculosis is fast becoming, in fact already is, a class disease; it is much more prevalent among the poor than the well-to-do. Hence the prevention of tuberculosis has become a sociological problem. Poverty with all its attendant hardships, such as poor food, bad housing, overwork, and worry, diminishes resistance to the infection; while prosperity, which buys good food, rest, change of air and scene, choice of occupation, and diversion, increases our resistance to the infection. An increase of wage or decrease in the cost of living; shortening the hours of work; improving the conditions of industrial hygiene; adding to the number of holidays; playgrounds, parks, and wholesome recreation, all help to increase our resistance against and diminish the prevalence of tuberculosis. Science has shown the way; it remains for society to apply the knowledge.

The Difference Between the Human and the Bovine Tubercle Bacilli. —There are at least three kinds of tubercle bacilli: human, bovine, and avian. The human and bovine varieties resemble each other closely; the essential difference lies in the fact that the human type is very pathogenic for man, but has little pathogenicity for cattle, rabbits, guinea-pigs, monkeys, and other animals. On the other hand, the bovine type is very pathogenic for almost all mammalian animals except man; it is pathogenic for man, but less so than the human bacillus. Even when large numbers of the human variety are injected into a calf, a general disease does not usually result; at most only a local lesion is produced. One one-hundredth of a gram of a pure culture of a bovine race injected subcutaneously is sufficient to cause generalized tuberculosis and death in a rabbit in about 6 weeks; while ten or a hundred times this quantity of a human strain produces at most a slight localized tuberculosis.

The human bacillus grows more luxuriantly upon culture media, covering the entire surface of the medium with a rich, dry, crinkled, mold-like vegetation. The growth of the bovine bacillus upon artificial culture media is more sparse, thinner, less extensive, and somewhat slower. According to Theobald Smith, who first pointed out the differences between these two types, the human bacillus produces more acid in artificial culture media and a different reaction curve than that produced by the bovine bacillus.

Morphologically the bovine bacillus is usually shorter, plumper, and stains more uniformly than the human bacillus, which is ordinarily club-shaped, irregular, and stains with interrupted markings. The morphological and tinctorial characters are not sufficiently distinctive to distinguish one type from the other.

It is doubtful whether there are any specific differences between the tuberculins of bovine and human origin.

The *avian tubercle bacillus* is found most frequently in chickens and also in pigeons, pheasants, and guinea-fowl. Geese and ducks appear immune. The avian bacillus is quite pleomorphic and stains somewhat more readily than either the human or bovine types. The avian bacillus grows luxuriantly upon artificial culture media at 45° C. and even multiplies at temperatures as high as 50° C., which is in marked contrast to the mammalian types, which do not vegetate above 40° C. The avian bacillus grows rapidly, so that upon glycerin-agar or upon blood serum there is an abundant growth in 10 days, which consists of a white, moist, and fatty mass quite different in young cultures from the dried and crinkled appearance of the human type. Guinea-pigs show a decided resistance to the avian cultures, but rabbits are susceptible. Chickens and pigeons may be infected with certainty by feeding, and it is probable that in nature avian tuberculosis is generally transmitted in this way.

Fish tuberculosis shows a marked difference to the races found in warm-blooded animals. The bacillus grows between 12° and 36° C., the optimum temperature being 25° C. It was first found in a carp and is pathogenic for frogs. Neither the avian nor the fish tubercle bacilli are pathogenic for man.

Bovine Tuberculosis in Man.—Concerning bovine tuberculosis in man we now possess definite knowledge which permits of precise statements. At one time the danger of bovine tuberculosis to man was greatly exaggerated. Koch went too far on the other side when he announced at London before the International Congress on Tuberculosis in 1901 that there was practically no danger of man contracting tuberculosis from cattle. In recent years Koch modified this dictum, for it was soon proven that the bovine bacillus has a certain amount of pathogenic power for man and that some of the tuberculosis in man is contracted from bovine sources. If only 1 per cent. of the deaths from tuberculosis in the United States were caused by bovine tubercle bacilli, it would mean 1,600 deaths yearly. It is now estimated that perhaps 7 per cent. of the tuberculosis in man is of bovine origin.

Pulmonary tuberculosis in man is practically never associated with the bovine bacillus. Bovine tuberculosis in man is usually a disease of the lymph glands—the lymph nodes of the cervical region and the lymph nodes in the abdomen being especially attacked. This is doubtless due to the fact that the portal of entry of the bovine bacillus is usually through the tonsils or the small intestines. Bovine tuberculosis may become a fatal infection in man when it is generalized through the blood in the form of acute miliary tuberculosis or when it localizes in the meninges or other vital parts. About one-quarter to one-half

of all cases of tuberculosis in children under 5 years of age is associated with the bovine type. It is probable that all these cases derive their infection through the tubercle bacilli in cow's milk. There is little danger from meat, as this is usually cooked, and tuberculosis of the muscles is exceedingly rare. Meat may become contaminated with tubercle bacilli as a result of unclean butcher's tools or unsanitary methods of handling, or from tuberculosis of attached glands.

The following table shows the relation between bovine and human tuberculosis in 1,040 cases. Six hundred and six of these cases were collected from the literature and include those studied by the English and German Commissions; 434 of the cases were studied in the research laboratory of the New York Board of Health by Park and Krumwiede:

TABULATION OF CASES EXAMINED AT THE RESEARCH LABORATORY, NEW YORK CITY DEPARTMENT OF HEALTH, BY PARK AND KRUMWIEDE

Diagnosis of Cases Examined.	Adults 16 Years and Over.		Children 5 Years to 16 Years.		Children Under 5 Years.	
	H.	B.	H.	B.	H.	B.
Pulmonary tuberculosis	278	..	8	..	5	..
Tuberculous adenitis, inguinal and axillary	1	..	4	..	..	..
Tuberculous adenitis, cervical	9	..	19	8	6	12
Abdominal tuberculosis	1	..	1	1	..	3
Generalized tuberculosis, alimentary origin	..	..	..	..	1	1
Generalized tuberculosis	2	..	1	..	12	4
Generalized tuberculosis including meninges	..	..	..	..	18	1
Tubercular meningitis	..	..	1	..	14	1
Tuberculosis of bones and joints	1	..	10	..	6	..
Genitourinary tuberculosis	3	1	1	..	..	..
Tuberculous abscesses	1	..	..	..	..	..
Totals	296	1	45	9	62	22

Total Cases, 426.

TABULATED SUMMARY OF CASES COLLECTED FROM THE LITERATURE

Diagnosis of Cases Examined.	Adults 16 Years and Over.		Children 5 Years to 16 Years.		Children Under 5 Years.	
	H.	B.	H.	B.	H.	B.
Pulmonary tuberculosis	290	1 (?)	3	..	7	..
Tuberculous adenitis, axillary	1	..	..	..	2	..
Tuberculous adenitis, cervical	13	1	14	12	9	8
Abdominal tuberculosis	14	3	6	6	6	10
Generalized tuberculosis, alimentary origin	6	1	2	3	12	9
Generalized tuberculosis	26	..	3	1	16	1
Generalized tuberculosis including meninges, alimentary origin	..	..	1	..	3	8
Generalized tuberculosis including meninges	4	..	7	..	27	..
Tubercular meningitis	..	..	1	..	..	1
Tuberculosis of bones and joints	17	1	16	1	15	..
Genitourinary tuberculosis	8	..	..	..	..	..
Tuberculosis of skin	1	..	1	..	1	..
Miscellaneous Cases:						
Tuberculous tonsils	..	..	..	..	1	..
Tuberculosis of mouth and cervical nodes	..	1	..	..	..	..
Tuberculous sinus	1	..	..	..	..	..
Sepis. latent bacilli	..	..	..	..	1	..
Totals	381	8	54	24	99	37

Mixed or Double Infections, 3 cases:

Generalized tuberculosis. Alim. Orig. 30 yrs. Human and bovine type in mesenteric node. Human type in bronchial node.

Generalized tuberculosis. Alim. Orig. 5½ yrs. Human type in spleen. Bovine type in mesenteric node.

Generalized tuberculosis incl. meninges. Alim. Orig. 4 yrs. Human type in meninges and bronchial nodes. Bovine type in mesenteric nodes.

Total Cases, 606.

From a study of these 1,040 cases we find:

16 years and over.....686 cases— 9 with bovine bacilli— 1.3%
Between 5 and 16 years.....132 " —33 " " " —25.0%
Under 5 years.....120 " —59 " " " —49.1%

Of 568 cases of pulmonary tuberculosis, none had the bovine bacillus. Cases under 5 years of age, 15 per cent.

It should be remembered that many of the cases included in the above total were selected cases. The 436 cases studied in the Research Laboratory in New York, however, were not selected; of these cases the following were found associated with the bovine bacillus:

Diagnosis	Adults	Five to Sixteen	Under Five
Pulmonary tuberculosis	None	None	None
Tuberculous adenitis, cervical	4%	37%	57%
Abdominal tuberculosis	16%	50%	68%
Generalized tuberculosis	3%	40%	26%
Tubercular meningitis with or without generalized lesions	....	....	15%
Tuberculosis of bones and joints	5%	3%	....

Since the above tabulations Park and Krumwiede [1] have collected a total of 1,511 cases which give the following:

PERCENTAGE INCIDENCE OF BOVINE INFECTION

Diagnosis.	Adults 16 Years and Over.	Children 5 to 16 Years.	Children Under 5 Years.
Pulmonary tuberculosis	.4%	0%	2.8%
Tuberculous adenitis, cervical	2.7%	38%	61%
Abdominal tuberculosis	20%	53%	58%
Generalized tuberculosis, alimentary origin	14%	57%	47%
Generalized tuberculosis	0%	16%	8.6%
Generalized tuberculosis, including meninges, alimentary origin	0%	0%	66%
Tubercular meningitis (with or without generalized lesions other than preceding)	0%	0%	4.6%
Tuberculosis of bones and joints	3.3%	6.8%	0%
Tuberculosis of skin	23%	60%	0%

As is evident from the table summarizing the total cases reported, many of those in children had slight or latent infections, found on their death from other causes. The percentages deduced, therefore, only give the incidence of infection, nothing more.

Weber, of the Imperial Board of Health of Germany, has made observations to determine just how much danger there is in drinking milk containing bovine tubercle bacilli. The milk coming from all known cases of udder tuberculosis was traced to the consumer and all the persons drinking such milk or using fresh milk products from in-

[1] *Jour. Med. Research,* XXVII, 1, Sept., 1912.

fected sources were examined with reference to tuberculosis. In all 113 separate investigations were made, including 628 persons (284 of whom were children, 335 were adults, and 9 of unstated age), all of whom had undoubted opportunities of consuming milk or fresh milk products from cows having tuberculosis of the udder. The evidence presented is not equally valuable in each investigation. In 44 of the 113 investigations cited, the milk was either heated, used in coffee or tea, or mixed with milk from apparently tuberculosis-free cows before it was consumed.

Three hundred and sixty persons (of whom 151 were children, 200 adults, and 9 of unknown age) were known to use milk or milk products, such as butter, buttermilk, sour milk, and cheese, which came from cows having undoubted tuberculosis of the udder. Of these 360 persons 2 were shown, by actual animal experimentation, to have infections with the bovine tubercle bacillus. Both positive cases were children with tuberculous neck glands. Six other children and 1 adult had glandular swellings in the neck, and in 4 other children and 1 adult there was a strong suspicion on the part of the attending physician that abdominal tuberculosis was present.

In another series of 360 persons, 12 children and 1 adult had swellings of the lymph glands of the neck. In this group the diagnosis was not confirmed bacteriologically.

Weber concludes from these studies that the danger which man undergoes through the consumption of uncooked milk and milk products of cows having tuberculosis of the udder is similar to the danger which persons having well-marked pulmonary tuberculosis exhibit for their fellowmen, although very much less. He believes it is fair to assume from the statistics presented above that the danger from drinking uncooked milk or using milk products of cows with tuberculous udders is surprisingly small.

Woodward voices the prevailing opinion when he maintains that the more deeply we go into the subject, the bovine side of the question comes to take a larger and larger place, especially in connection with surgical and abdominal tuberculosis, not only in the child but even in the adult.

From the standpoint of our present knowledge we must consider that practically every case of bovine tuberculosis in man is ingestion tuberculosis, contracted from milk or fresh milk products. How the tubercle bacilli get into milk and the frequency with which it is infected are discussed on page 513.

Occasionally butchers and also pathologists at autopsies become infected with the bovine bacillus through wounds. These accidents furnish further experimental proof that the bovine type of the tubercle bacillus possesses a certain degree of pathogenicity for man.

MODES OF INFECTION

There are two great sources of human tuberculosis: the principal source is man himself; the secondary source is cattle.

From man tubercle bacilli leave the body mainly in the sputum, where they are found in great numbers in all open cases of pulmonary tuberculosis. Tubercle bacilli may also leave the body in the discharges from any open tuberculous lesion wherever situated, especially in discharges from the lymphatic glands, bones, intestinal or genitourinary tracts, or the skin. In pulmonary tuberculosis some of the sputum is swallowed and the bacilli appear in the feces, therefore any or all of the discharges from the body may be infective. But, from the practical standpoint of prevention, the bacilli in the matter brought up from the lungs is the source of the danger in the overwhelming majority of cases.

Practically all observers agree with Koch that human sputum is the main source of human tuberculosis. Whether the tubercle bacillus is usually transferred directly or indirectly, in moist or in dry state, by inhalation or ingestion, are questions still undetermined. The question at issue is a quantitative one; that is, how often are we infected by the direct aerogenic route, how often through the tonsils and upper respiratory passages, how often through the digestive tube, etc.?

Aerogenic Infection.—The belief that tuberculosis is air-borne, that is, that pulmonary tuberculosis is a primary inhalation tuberculosis, has long been the natural and favorite theory, from the fact that the lungs are most frequently affected. This opinion was strongly expressed by Koch in 1884, and repeated by him in 1901, at the British Congress on Tuberculosis. For many years it found practically universal acceptance. Cornet taught that the tubercle bacilli entered the lungs in the dust of dried and pulverized sputum.

The evidence of pathologic anatomy strengthens the belief in the importance of aerogenic infections as the chief portal of entry. Thus, the recent studies by Ghon,[1] at the St. Anne's Children's Hospital in Vienna, indicate very strongly that the actual path of infection is by the aerogenic route. Approximately 95 per cent. of 184 autopsies studied by him represent a primary localization of the bacilli in the lungs. On the other hand, it seems that direct aerogenic infection has been greatly overestimated, and some students of the subject go so far as to state it is of little or no practical importance. It is supposed that very few bacteria suspended in the air actually reach the lungs, being caught on the moist mucous membranes of the upper air passages. Further, tuberculosis of the lungs is usually at the apex, which is not in the direct line that floating particles in the air would usually be

[1] "Der primäre Lungenherd bei der Tuberkulose der Kinder," Berlin, 1912.

mechanically carried. It is true that dust under certain conditions may contain tubercle bacilli, but it is now known that this organism soon dies when exposed to the sun and air, and that the dust out of doors is not apt to contain the live bacilli, and when it does so the dilution must be enormous. It is different with house dust. Tubercle bacilli may live a long time in dark, moist places, but even here the danger cannot be as great as might be supposed when we study the nature of tuberculous sputum. This substance is usually tenacious and gummy, and dries into tough, glue-like masses, which are pulverized with great difficulty. It therefore seems unlikely that dust under ordinary circumstances would contain dangerous numbers of live tubercle bacilli. The danger from this source is further diminished when we consider that a large number of tubercle bacilli die in sputum even when protected from sunlight and other injurious influences. It is now known that even under most favorable conditions in artificial culture media the great majority, perhaps 99 per cent., of the bacilli die within three months. Transplants made from cultures over three months old usually do not grow. The danger of house dust containing live tubercle bacilli from a quantitative standpoint is, therefore, reduced.

It is quite possible that the first infection does not produce the disease; that is, when a few tubercle bacilli land upon the lungs the tissues do not react and the bacilli are carried to the bronchial lymph glands. This first infection, however, sensitizes the parts, so that the second time the bacilli lodge the tissues react vigorously and a local lesion may result. A dusty atmosphere, even though it contains no tubercle bacilli, is, however, exceedingly dangerous, in that it irritates the delicate mucous membranes and thus opens the door for infection.

One point of importance in this controversy is the experimental evidence that it requires very few tubercle bacilli by inhalation to produce the disease, whereas it requires hundreds and even thousands to cause intestinal infection. This is given as a reason why infection via the digestive tract is comparatively rare in man, for he fortunately would seldom receive the necessary numbers of human bacilli by the mouth.

Cornet and others have actually found live tubercle bacilli in the dust and upon objects of rooms where tuberculous patients are careless with their sputum. In one of Cornet's experiments 47 out of 48 guinea-pigs exposed to the dust produced by sweeping a carpet with a stiff broom became tuberculous. The carpet had been purposely infected with tuberculous sputum shortly before. Dust containing tubercle bacilli may also enter the atmosphere from soiled linen, upholstery, handkerchiefs, and other fabrics containing the dried tuberculous sputum. Tuberculous dust may also be stirred up by walking over floors and the dragging of the infection by ladies' skirts.

Droplet Infection.—When it was found that the danger from dust theoretically was not as great as was supposed, Flügge called attention to the fact that in speaking, coughing, sneezing, and in other violent expiratory efforts the fluid contents of the mouth are sprayed into the air in the form of a fine mist. These tiny droplets contain tubercle bacilli or germs of any other infection that may be in the mouth. Ordinarily these droplets are carried several feet, but under exceptional circumstances may be carried 30 or 40 feet or more; however, at these distances the dilution is enormous and the danger, therefore, much diminished. The tubercle bacilli contained in the droplets sprayed from the mouth are fresh and virulent, and may land directly upon the mucous membranes of the healthy individual or may be conveyed indirectly through food, fingers, and other objects. There is danger from droplet infection, but it cannot be the usual mode of transmission in tuberculosis from the nature of the circumstances. The danger from droplet infection is increased by close association with the patient in stuffy, ill-ventilated rooms, especially if the individual does not take proper care in coughing and sneezing. For a further discussion of droplet infection see page 632.

Ingestion Infection.—Little by little the view gained ground that some cases of tuberculosis, particularly in children, might be due to bacilli entering through the mucous membrane of the alimentary canal. Now we recognize that much of the tuberculosis in children comes through the alimentary tract. Many years before the discovery of the tubercle bacillus Chauveau (1868) was inclined to the belief that the alimentary canal may be the portal of entry in tuberculosis. Woodward in 1894 maintained that the infecting bacilli might reach the lungs through some part of the alimentary canal. He drew attention to the fact that in many children, and also in animals fed on tuberculous material, the lungs may be markedly affected. He traced the course of the infection through caseous or old calcareous mesenteric glands up through the diaphragm to the posterior mediastinal glands, and so to the lungs. Still in 1899 analyzed 259 fatal cases of tuberculosis occurring in the Hospital for Sick Children, London, and concluded that the infection had occurred through the alimentary canal in 20.5 per cent. of the cases. Shennan in 1900, dealing with 316 autopsies at the Royal Hospital for Sick Children in Edinburgh, found this ratio to be 28.1 per cent.

There is no doubt that the lungs are more or less involved in all cases of generalized infection, especially in children, but these are not cases of pulmonary tuberculosis (phthisis) in the usual meaning of the term. It is phthisis or pulmonary tuberculosis which causes 70 per cent. of all the mortality from tuberculosis and whose mode of origin is now in question.

Behring in 1903 maintained that the tubercle bacilli might be taken up from the intestine and pass through the mesenteric glands, so gaining access by the blood stream to the lungs without leaving any lesion in the gut or glands to mark the portal through which they had entered or the route by which they had traveled, and that pulmonary tuberculosis was commonly caused in this way. Behring's theory of the origin of phthisis did not find a ready acceptance. Nevertheless, the belief that phthisis may be caused by bacilli which have been swallowed and absorbed from the digestive tube gradually gained ground. Vallée in 1904 concluded from his own investigations that ingestion of dust or food infected with tubercle bacilli was the quickest and surest method of infection. A little later Calmette (1905) of Lille appeared as a strong supporter of this view. Calmette went so far as to assert that the immense majority of cases of pulmonary tuberculosis in man are caused by ingested bacilli and not by inhalation. Whitla, in 1908, and Symmers repeated some of this work and became converted to Calmette's doctrine, and these views have gained a number of adherents. Cobbett (1910) considers that the ingestion theory is based on a slender substructure of experiments from which too sweeping conclusions have been found. Thus Calmette and his colleagues claim that even anthracosis is caused not by the carbon particles inhaled, but the particles ingested, which pass through the intestinal mucosa and lodge in the lungs. Cobbett showed the experimental error and demonstrated that India ink intimately mixed with cream is not absorbed in any great amount from the intestine, for the cream reappears of a normal color in the lacteals. He found, however, that feeding finely divided carbon matter caused traces of pigmentation in the lung and bronchial glands when long continued. Heller and Vulcanstein showed that the feeding of large amounts of coal dust never produces that grade of anthracosis which is found after the inhalation of much smaller amounts.

There is now sufficient proof to state definitely that tubercle bacilli, when taken in food or drink, may pierce the mucous membrane of the digestive tube and produce lesions in distant parts of the body. It is also demonstrated that the tubercle bacillus may thus travel without leaving macroscopic evidence of its passage in its wake. Fraenkel[1] and others have shown that the tubercle bacilli may pass through the uninjured skin of guinea-pigs, leaving no trace of their passage at the place where they had rubbed upon the skin, but causing tuberculosis of the internal organs. Ravenel and others have shown that tubercle bacilli may pass through the intestinal wall without leaving a trail behind them. It does not, therefore, necessarily follow that the seat of

[1] *Hyg. Rundschau*, XX, 15, Aug. 1, 1910, p. 817.

the primary lesion in tuberculosis is the site of the entrance of the infection.

It is also claimed that, no matter how the tubercle bacillus reaches us, whether in dust or droplets, by kissing or through fingers, flies, cups, handkerchiefs, or milk, it either passes through the tonsils or mucous membrane of the upper respiratory passages, or is carried into the intestinal tract and absorbed from the intestines. Viewed in this light, the portal of entry even in dust infection may be through ingestion rather than through direct aerogenic infection of the lungs. Experimentally it is easy to prove that tubercle bacilli given by the mouth may produce a generalized and fatal tuberculosis; thus, of 100 guinea-pigs given one large feeding of a bovine culture by Rosenau and Anderson, 99 died of tuberculosis. That infection by ingestion does not tell the whole story is judged from the fact that primary tuberculosis of the mesenteric nodes in man is not as common as we might expect. On the other hand, it is claimed that the tubercle bacillus may pass these lymph glands, leaving little or no trace behind them. Thus the work of Weichselbaum and his pupils, Bartel, Neuman, and Spieler, strengthens the importance of ingestion as the portal of entry. These investigators found that the tubercle bacillus produces, in addition to the specific tubercles, other lesions of a simple lymphatic hyperplastic character. These early lesions are called the "lymphoid stage" ("lymphoide stadium"). The recognition of this early stage is of importance in determining the point of invasion. The evidence obtained from the macroscopic appearance of the lesions at autopsy must be supplemented by microscopic studies. Bartel and Spieler found that in ingestion experiments the different lymphatic groups were infected with the following frequency, judged by the lymphoid stage:

Tonsils and surrounding	11.7 per cent.
Cervical glands	58.8 per cent.
Bronchial glands	52.9 per cent.
Mesenteric glands	100.0 per cent.

These investigators assume that the tubercle bacillus is carried from the mesenteric or the neck glands either through the lymphatics directly or through the thoracic duct and the arterial circulation to the lungs and other tissues and organs of the body. The disease usually localizes itself in the lung because this organ presents the least resistance.

Weichselbaum believes that ingestion tuberculosis occurs much more often in man than is commonly supposed and especially in children. He assumes that the tubercle bacilli may pass through the mouth, nose, or throat. It seems immaterial whether the bacillus is taken with food

or other substances placed in the mouth, or is contained in the inspired air, or enters the mouth and nose through any other medium. The first lesions do not consist in the formation of specific tubercles, but in the so-called lymphatic tuberculosis. This stage lasts a variable time and may end in recovery or may lead to specific tuberculosis either through reinfection, or it may light up itself without a new infection. The specific tubercles may occur either at the portal of entry or in the lungs and bronchial glands or in other organs.

Behring (1903) brought forward the theory that alimentary infection occurs in the early months of life. The tender mucous membrane of babies permits the bacillus to pass readily. The bacilli remain latent in the tissues and acquire increased activity later in life. According to this view tuberculosis of adults is the "end of a song, the beginning of which for the unfortunate patient was sung in the cradle." If this view were correct, the majority of cases of tuberculosis in adults would be associated with the bovine bacillus, unless the bovine bacillus has the power of changing to the human type during its long stay in the body. This is not likely.

It is clear from the evidence at hand that pulmonary tuberculosis may arise either by inhalation or by ingestion. The problem for us now to solve is a quantitative one; that is, what percentage of cases are air-borne and what percentage come through the mucosa of the digestive tract? Opinions differ widely, but opinions are of little value. We must have the facts before we can give the final answer to this very important and practical question.[1]

Flies.—Under certain circumstances flies may readily transfer tubercle bacilli from exposed sputum to fingers, lips, or food. This may account for an occasional case.

Water.—Large quantities of tuberculous sputum that escape disinfection and an additional large number of tubercle bacilli in the excreta finally reach the drinking water. The tubercle bacillus is particularly resistant to putrefactive processes, and may live a long time in water. The use of contaminated water can, therefore, not be disregarded. A study of the vital statistics of Hamburg, Lowell, and Lawrence seems to show a diminution in tuberculosis following a purification of the water supply by filtration (Mills-Reinke Phenomenon, page 804).

Contact Infection.—The majority of cases of tuberculosis contract the disease through "contact." Contact infection is a general and convenient term; it implies the rather quick transference of fresh infection in which the bacilli pass from one individual to the other in a brief space of time and through a short distance. Contact infection

[1] An exhaustive and able summary of this question will be found in Bulloch's article in Allbutt's "System of Medicine," from which some of the facts in this article have been used.

may be either direct or indirect; through dust, through bacilli in the air, or through contaminated food, through soiled fingers or objects; through flies, as well as in numerous other ways. The infections transferred through kissing, pencils, pipes, toys, cups, and other objects all come under the convenient category of "contacts." Even the infection through droplets is included in the present-day conception of contact infection. The term is a practical one, and implies close association, though not necessarily actual contact, between the sick and the well. Viewed in this sense, tuberculosis is a house disease or a family disease. With this conception it makes little practical difference whether the infection enters the body through the respiratory tract or the digestive tube. Either or both would be possible in regarding the disease as contagious in the sense of contact infection.

Although there is some doubt concerning the exact mode of transmission and the portal of entry that the tubercle bacillus usually takes, we have sufficient knowledge to guide our preventive measures with every assurance of success. One thing is certain: tuberculosis is an infection spread mainly from man to man, usually through direct association between the sick and the well; and secondarily from cows, through milk.

IMMUNITY

Man possesses a considerable resistance to tuberculosis. This is shown by the fact that many cases recover spontaneously and that perhaps all individuals who reach the age of 30 years and who spend most of this time in association with their fellowmen under the usual urban conditions have at one or more times been infected. The resistance to tuberculosis increases after middle life, due perhaps to the immunity which is induced by these prior infections. There is probably no true racial immunity to tuberculosis. Some races show a smaller incidence to the disease, owing probably to modes of life, habits of nutrition, and conditions of exposure.

The human organism is capable of taking care of a certain amount of infection. The dose, that is, the number, of tubercle bacilli and their virulence, is, therefore, a very important factor in determining infection. This may readily be demonstrated upon susceptible animals and is doubtless true of man. Frequent reinfections occurring at short intervals with small numbers of tubercle bacilli doubtless break down the immunity. In man the balance between immunity and susceptibility to tuberculosis is delicately adjusted: there is a very small factor of safety. The resistance to the infection may be increased by attention to personal hygiene, fresh air, and good food; immunity may readily be broken down by any weakening influence; herein lies the keynote of personal prophylaxis.

The immunity to tuberculosis is not sufficiently strong to overcome a large amount of infection. As in all other infectious processes, the strongest and most robust individuals in the prime of life succumb to the disease in a short time if they receive into their system a large number of virulent tubercle bacilli. Hence the avoidance of the infection is one of the most important of our preventive measures.

The mechanism of the immunity to tuberculosis is probably exceedingly complex. There is no antitoxic immunity. The tuberculins are not true toxins. Phagocytosis and cellular reactions play a very important rôle. The recent studies upon anaphylaxis throw a certain amount of light upon the mechanism of immunity in tuberculosis. The phenomenon of hypersusceptibility is beautifully illustrated in the action of tuberculin, which is a comparatively harmless substance to a normal individual, but produces a marked reaction in a sensitized individual. This reaction must be useful in protecting the organism against the invasion of the tubercle bacillus, and also in guarding it against the spread of the disease after it has become localized. Thus, if tuberculin is placed upon a normal conjunctiva no reaction follows.[1] This first application, however, sensitizes the tissues of the conjunctiva so that, if the application is repeated after the lapse of a few weeks, there is a violent reaction. The same phenomenon doubtless occurs when a tubercle bacillus lodges in a lymph gland or in the lung or some other part of the body. The first time it meets with little resistance; the next time the tissues react immediately and vigorously. All of nature's protecting agencies, such as the germicidal substances in the blood, the phagocytic cells, and antibodies, are concentrated upon the point where they are most needed. In the same way the body protects itself against the extension of a tuberculous focus. The parts surrounding a tubercle become sensitized and react so as to encapsulate the focus with a cellular and fibrous coat of mail. This reaction is probably stimulated by small amounts of tuberculin produced within the tuberculous focus. When the tuberculin is not produced autogenously in sufficient amounts, as in chronic lesions of the bones, or inactive processes of the glands or skin, the specific reaction may be stimulated to advantage by the injection of small quantities of tuberculin. If, however, the tuberculin is given in too large amounts or too frequently, the power of reaction is readily broken down. When this occurs the mechanism of immunity has been destroyed, there is little resistance left to the extension of the infection, and death soon occurs. Clinical experience has demonstrated the danger of large doses of tuberculin or small amounts too often repeated in tuberculosis. The same may readily be demonstrated experimentally in the lower animals. These facts are of fundamental importance in the use of tuberculin.

[1] Rosenau and Anderson, *J. A. M. A.*, Vol. I, March 28, 1908, p. 961.

It is quite proper to deny dogmatically the hereditary transmission of tuberculosis in educational pamphlets for popular use. The infection is not transmitted hereditarily, although it occasionally passes from mother to fetus congenitally. Tubercle bacilli do not occur in the spermatozoon, and do not appear in the seminal fluid. They are not found in the ovum; in fact, a tubercle bacillus in the ovum would doubtless result in the death of the egg. The bacilli, however, may pass from mother to fetus through the placenta. Warthin shows that placental tuberculosis is more common than is supposed. The lesions in the placenta are not those of typical tubercle formation.

While the tubercle bacillus itself is rarely transmitted from parent to offspring, an hereditary tendency or disposition to the disease may be transmitted. We have no definite knowledge as to what this decreased resistance consists in; it may be a diminished power of reaction. For this view there is analogy in the experiments upon anaphylaxis in guinea-pigs, in which it has been shown that hypersusceptibility to a foreign protein such as tuberculin may be transmitted from mother to young.

A mild infection with bovine tuberculosis in early life seems to leave a certain degree of immunity against the human strain. At least children who have glandular tuberculosis of the bovine type in childhood are said to be less apt to have tuberculosis of the lungs in later life. Likewise, the human strain injected into cattle produces a definite immunity against the bovine type. Cattle are now immunized by the intravenous injection of 2 c. c. of a suspension of a pure culture of the human tubercle bacillus. This produces an immunity which probably lasts for 1 to 2 years. It should be remembered that the human bacillus under these circumstances remains alive for a very long time, and may appear in the milk provided there is a lesion in the udder. This presents a danger which cannot be disregarded.

Trudeau long ago showed that the only definite immunity that could be induced in experimental animals was through the use of live tubercle bacilli. Webb and Williams[1] have produced a certain amount of immunity in guinea-pigs and monkeys by the injection of live tubercle bacilli. The first injection consists of the introduction of a few bacilli (from 1-200), which is repeated subcutaneously at varying intervals. Two children have also been successfully "vaccinated" with upward of 600 virulent human tubercle bacilli without infection being produced.

RESISTANCE OF THE VIRUS

We have no easy method of determining just when the tubercle bacillus dies. The criterion of death depends upon animal experimentation.

[1] "Immunity in Tuberculosis," *J. A. M. A.*, Oct. 28, 1911, Vol. LVII, No. 18, p. 1431.

The tubercle bacillus has no spore and may be classed with other non-spore-bearing organisms so far as its viability is concerned. Its virulence fades before it dies. It is doubtful whether the waxy substances protect the bacillus against external harmful influences to any unusual extent. The thermal death point is 60° C. for 20 minutes. This is much less than was once considered.[1] Failure to recognize the lesions produced by the dead tubercle bacillus is responsible for some of the false conclusions reached by experimenters upon this subject.

From a practical standpoint the resistance of the tubercle bacillus in sputum is of prime importance. Protected from the sunlight it is now known that they may live in dried sputum for months. All the bacilli do not survive under these conditions, but we lack methods to determine the quantitative reduction.

The tubercle bacillus withstands cold very well. It has a marked resistance against putrefactive processes. It will live a year in water, which is a fact not to be neglected, as many tubercle bacilli finally find their way into drinking water, and infection through this source is possible.

For the destruction of the bacilli in sputum only very strong germicides or exposure to steam or boiling water should be depended upon. Five per cent. carbolic acid is sufficient, provided equal parts of sputum and solution are mixed and the exposure continued for 24 hours. Ten per cent. lysol acts in 12 hours. Bichlorid of mercury is not applicable for sputum disinfection, as it cannot penetrate the albuminous mass. Formalin, 10 per cent., may be used.

Sunlight is one of the best germicides and often destroys tubercle bacilli quickly. In direct sunlight the bacilli die in a few hours, in diffuse sunlight in a few days, provided the sputum masses are not too thick.

Antiformin is a differential germicide, killing most non-spore-bearing bacteria, but acting more slowly upon the tubercle bacillus. Antiformin is a strongly alkaline solution of sodium hypochlorite. (Page 1020.)

PREVENTION

Preventive measures are based upon two important facts: that tuberculosis is an infection mainly spread from man to man through direct association, and secondarily from cattle through infected milk. Preventive measures fall into two categories: (1) avoiding the infection, and (2) increasing resistance through personal hygiene. Both are necessary. The infection may be avoided through segregation; the use of milk from tuberculin-tested cattle, else pasteurized; education;

[1] The thermal death point of pathogenic microörganisms in milk. M. J. Rosenau, Hyg. Lab. Bull. U. S. Pub. Health and Mar. Hosp. Serv., No. 42.

disinfection; proper disposal of tuberculous sputum; the avoidance of contact with open cases, especially with those who do not use proper precautions; early diagnosis, etc. Increased resistance may be gained through fresh air, good food, rest, and compliance with the dictates of personal hygiene. This part of the subject includes sociologic and economic reforms, without which the warfare against tuberculosis cannot succeed. Improvement in housing conditions, lowering of the cost of living, increase in the scale of wages, and all forms of uplift help secondarily to diminish the amount of the disease. Furthermore, it will be necessary to consider secondary agencies, as preventive clinics, industrial insurance, notification, open-air schools, day and night camps, etc.

It is well to remember that tuberculosis has gradually declined in England and also in Massachusetts since about 1850—before the tubercle bacillus was discovered.

Segregation.—Tuberculosis is a "contagious" disease, and it is now perfectly plain that one of the most important single preventive measures in this as in all other communicable diseases consists in isolation. A case isolated is a case neutralized, hence the great value to the community of sanitarium treatment. Isolation in this case refers only to those individuals having tubercle bacilli in their sputum, and especially to the advanced and helpless cases. The isolation in tuberculosis need not go to the extreme practiced in the acute communicable fevers. In fact, we cannot for many years to come object to giving a case of open pulmonary tuberculosis his complete liberty, provided he is careful and cleanly and uses proper precautions in the disposal of his expectoration. When the disease becomes less prevalent more stringent and arbitrary measures may then be enforced.

"Every case of tuberculosis isolated means an average of at least three less new infections." Sanatoria should, therefore, be attractive and as cheap as it is possible to run them. Free hospital care for the incurable cases is necessary, especially for the poor. Tuberculosis has diminished most in those countries where sanatoria are most in use.

Personal Prophylaxis.—Personal prophylaxis consists in avoiding the infection and in obeying all the dictates of personal hygiene—that is, living a clean, normal, and temperate life.

Close association with persons known to have tubercle bacilli in their sputum is hazardous. This becomes especially dangerous when the contact is prolonged and intimate, such as working in the same room, especially if it is small and ill-ventilated, or sleeping in the same bed. The more intimate the association and the less care the tuberculous individual takes with the expectoration, the greater is the danger. The infection may further be avoided by refusing to drink from common cups, by taking care in placing objects to the mouth that do not belong there, by avoiding dusty atmospheres, and refusing to

drink milk that does not come from tuberculin-tested cattle unless it is pasteurized.

Mechanical obstructions to breathing should be corrected, by surgical methods if necessary. Functional lack of proportion in the chest and lungs of young people favor infection, and every effort should be made to help the child to outgrow them. Breathing exercises and outdoor play are especially useful.

A generous diet is one of the best prophylactics against tuberculosis. A fat-rich food favors the development of a water-poor body, and it is known from experimental observation that animals with the largest proportion of water in their tissues yield to infection more readily than others.

Resistance to the disease is increased by rest, fresh air, good food, sunshine, the avoidance of all depressing influences, such as worry, overwork, intemperance, and excesses of all kinds. Attention should be given to slight colds and other conditions known to be predisposing causes to the disease.

Education.—The prevention of tuberculosis, like all other widespread infections, depends for its success upon the education of the people. We are now in possession of sufficient information of a precise nature to place the facts in plain words before the public. This has been done in numerous excellent pamphlets and popular articles in the daily press and magazines, through lectures, exhibits, and meetings, so that there is now a widespread and correct understanding of the problem. The modern message in tuberculosis has been one of hope, in that the disease is curable; and one of fear, in that it is transmissible. The former has been a great encouragement and has added strength to the movement; the latter is also helpful, although it has run to extremes in some quarters. An unwarranted fear of tuberculosis (phthisiophobia) has subjected the tuberculous individual to severe hardships by branding him as a leper. Even cured cases of the disease now find difficulty in obtaining work. A wholesome regard for the infection is useful and helpful in preventive medicine, but an hysterical fear of tuberculosis is quite as unwarranted as a total disregard for the infection.

Notification.—Tuberculosis should be included among the list of diseases requiring compulsory notification. Without this important feature preventive measures are handicapped. The objection to compulsory notification is based largely upon sympathy with the large number of individuals affected and the sensitiveness of the afflicted. Compulsory notification may result in unnecessary harm, in that the knowledge of the fact may result in loss of occupation and an avoidance by his fellowmen on account of the fear people now have of associating with a tuberculous individual. These effects may, for the present, be

neutralized by considering the records as confidential communications between physician and health officer.

Tuberculosis is required to be reported in Maine, Michigan, Massachusetts (since 1907); many cities: Alameda, California; Asbury Park, N. J.; Boston, Buffalo, Cincinnati, New York, Salt Lake City, Trenton, Yonkers—also in Washington, D. C., Minneapolis, San Francisco, and Syracuse. The list is growing and the returns are gradually improving.

Disposal of the Sputum.—As the tuberculous sputum is the principal source of the infection, it should be disinfected or disposed of so that it will be harmless to others. Perhaps the best way is to receive the expectorated matter into cloths, which may be burned, or the material may be received into one of the various forms of sputum cups and finally burned or disinfected. Persons with pulmonary tuberculosis must be warned against the possible danger to others of coughing without holding the handkerchief before the mouth and nose; under no circumstances should they spit upon the floor. Penalty for spitting upon the sidewalk, upon the floor of public buildings, and in street cars serves a useful purpose in diminishing the spread of tuberculosis as well as other diseases.

In sanatoria and hospitals the infected material may be burned or disinfected with steam under pressure in a special autoclave, or disinfected with phenol (5 per cent.), lysol (2 per cent.), tricresol (2 per cent.), or formalin (10 per cent.).

Disinfection.—Rooms occupied by tuberculous individuals should be kept clean and disinfected from time to time. A thorough disinfection and cleansing should also be practiced before such rooms are occupied by other persons. This may be accomplished by mopping surfaces with the usual solutions of bichlorid of mercury or one of the coal-tar preparations, followed by formaldehyde fumigation and a mechanical cleansing, and then a thorough airing and sunning.

Early Diagnosis.—Early diagnosis plays an important rôle in successful prevention; not only does it give the individual the best chances of cure, but at the same time it assures the possibility of maximum protection to others. Through the use of tuberculin and through refinements of clinical methods it is now possible to diagnose tuberculosis at a stage when it was formerly not suspected. It is a great mistake, from the standpoint of prevention, to wait until tubercle bacilli appear in the sputum before making a diagnosis of tuberculosis. Probably many cases of "a slight run-down condition," of transient and irregular febrile attacks, are due to a small focus of tuberculosis hidden from the ken of the clinician. In such cases a course of rest, fresh air, and better food, with a change of scene, may often prevent irreparable damage. The establishment of preventive clinics to look after such cases and the maintenance of medical clinics to diagnose and

care for the early cases are important adjuncts to preventive measures.

Housing Conditions.—It has long been realized, even before the reasons were understood, that improvement in housing conditions diminishes the incidence to tuberculosis. This is a common observation in the stabling of cattle as well as the domicile of man. The reasons why improving the housing conditions diminishes the spread of tuberculosis are complex. In addition to raising the standard of living, better houses diminish the chances of contact infection, afford better air and more sunshine, and tend generally to the well-being and uplift of mankind. Municipalities do well to enact and enforce stringent laws regulating the construction of houses, offices, stores, and workshops. The congested and squalid slums are both a disgrace and a menace. Germs are social climbers, and many a palace is invaded with an infection from a nearby neglected alley. Philanthropists cannot do better than assist in improving the housing conditions of the poor.

Bovine Tuberculosis.—The prevention of bovine tuberculosis consists simply in using milk, cream, and fresh milk products from tuberculin-tested cattle. The cattle should be tested frequently; at least twice a year, for the disease may develop in the cow in a few months. When milk is used from non-tested cattle, it should be pasteurized, and the same precaution applies to the milk used for making cream, butter, ice-cream, and other fresh milk products.

Industrial Insurance.—Industrial insurance patterned after the plan used in Germany is a useful adjunct in the fight against tuberculosis. The German industrial associations under government supervision do more than care for the tuberculous workman. The heavy drains upon the funds of the industrial associations have been checked by the establishment of "preventoria." These are attractive country places where the working man can go when he is "run down." This simple measure is a great boon, and prevents the development of many a case of tuberculosis as well as other diseases.

Day camps, night camps, visiting nurses, and similar agencies are all helpful. In addition to the direct benefits, they teach the patient how to prevent the spread of the infection, how to sleep out of doors and its benefits.

The prevention of tuberculosis is no longer a medical problem—rather a sociological problem. The battle against tuberculosis has been waged with enthusiasm and the results are encouraging. Its eradication will, however, take a long time on account of the chronic nature of the disease and its widespread prevalence. We should be satisfied if we diminish the amount of tuberculosis appreciably in a generation. The momentum thus gained will increase rapidly. The time will then come when the comparatively few cases left may be treated by compulsory isolation or other aggressive measures. Persistence along the lines now

understood will in time control the disease, which will be the crowning achievement in preventive medicine.

DIPHTHERIA

Our knowledge of diphtheria is most satisfactory in that we know the cause of the disease and its modes of transmission; we are able to check its spread, and possess a specific preventive and curative agent of great potency.

Diphtheria spreads slowly from person to person and from community to community. It is not necessary to consider it endemic in special indigenous foci, because it is seldom completely absent in any large community. Newsholme points out that diphtheria epidemics and pandemics occur cyclically. The intervals between the years of epidemic prevalence vary greatly. In Boston diphtheria was epidemic in 1863-64, 1875-76, 1880-81, 1889-90, and 1894; in New York in 1876-78, 1880-82, 1886-88, and 1893-94; in Chicago in 1860-65, 1869-70, 1876-79-81, 1886-87, and 1890. The causes of these epidemic outbursts are not clear. They may be due to a fortuitous combination of such circumstances as a new crop of susceptible children, a particularly virulent strain of the bacillus, the opening of the schools, and similar factors favoring the spread of the infection. On the other hand, external conditions, such as dryness, may be important, for "diphtheria only becomes epidemic in years in which the rainfall is deficient. There is no instance of a succession of wet years in which diphtheria was epidemic." It is more than likely that the great outbreaks are due to a combination of the three factors—man, the bacillus, and the environment. Just as a spark in a forest may cause a brush fire or a conflagration, depending upon the amount of vegetable growth, its distribution, its condition as to dryness, the direction and force of the wind, the topography and nature of the soil, and a thousand and one other conditions, so diphtheria and other infections will smolder or burst into flame, depending upon many factors.

Diphtheria is said to prevail more in rural than in urban districts. Sir George Buchanan first pointed out that it has always displayed a more marked tendency to prevail in sparsely settled districts than in centers of population, although outbreaks in congested centers, schools, camps, on board ships, and in other crowded places, are common. In the tropics diphtheria is practically absent. Newsholme pointed out that it is more of a continental than an insular disease.

The fatality from diphtheria has been greatly lowered since 1904, owing to the use of antitoxin and owing also to refinements of diagnosis, as a result of which many mild cases are now included that were formerly omitted from the statistical records. Whether or not there has been a

natural tendency for the disease to become milder in recent years cannot be stated.

Diphtheria reaches its maximum prevalence in the autumn of each year, which corresponds to the seasonal prevalence of scarlet fever.

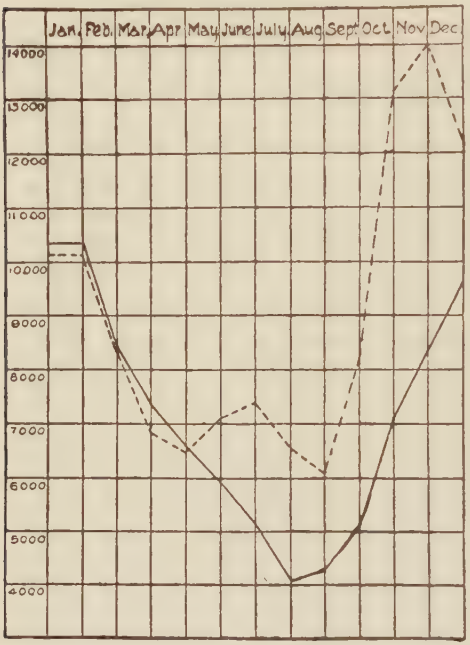

FIG. 16.—CHART COMPUTED FROM THE UNITED STATES CENSUS REPORT TO SHOW HOW THE OPENING OF THE SCHOOLS IN AUTUMN INCREASES DIPHTHERIA.

The broken line shows the number of cases among school children five to fourteen years old during 1900–04 in the registration area of the United States. The unbroken line shows the number of cases among children, from birth to five years of age, for same period and area. On this chart the augmented increase in diphtheria among school children from five to fourteen years of age, as compared with children under five years, is strikingly shown. (Mass. State Board of Health, *Monthly Bull*, Sept., 1910.)

In 1878 Dr. Thrushfield published papers illustrating the way in which diphtheria hung about damp houses. A damp dwelling favors sore throats and colds, and may thus open a way for invasion of the bacilli, just as any depressing influence may predispose to the infection. Children with scarlet fever or measles are especially prone to take diphtheria if the infection is around. Formerly imperfect drains and sewer gas were given as the causes of diphtheria; this is a fetish which dies hard.

Modes of Transmission.—The diphtheria bacillus almost always enters by the mouth or nose, and the lesions are usually localized in the mucous membranes of the throat, nose, larynx, or upper respiratory tract. The bacillus leaves the body in the discharges from the mouth and nose. Diphtheria occasionally affects other mucous membranes or abraded surfaces, such as the conjunctiva or vaginal mucous membrane, or open wounds, in which case the discharges from these lesions contain the infection.

The bacillus may be transmitted directly from one person to another, as by kissing, or exposure to droplet infection in coughing, speaking, and sneezing; or the infection may be conveyed indirectly from one person to another in a great variety of ways; most common among children, perhaps, are toys, slate pencils, food, fingers, handkerchiefs,

or other objects that have been mouthed first by the infected child and then by the susceptible child. Experience points clearly to the conclusion that diphtheria infection is transmitted usually by direct exchange of the flora of the nose and throat, rather than through inanimate objects.

Bacillus carriers play a large rôle in spreading the infection. Milk and other foods may become infected and transmit the disease.

The diphtheria bacillus is frail and soon dies when dried or exposed to sunlight, therefore air-borne infection is probable only in the case of close association, that is, within a few feet of the infected person and within the radius of the possibility of droplet infection.

The following description by Chapin illustrates how diphtheria and all other infections contained in the secretions from the mouth and nose may be transmitted; it also emphasizes the importance of education in personal hygiene based upon habits of biological cleanliness:

"Not only is the saliva made use of for a great variety of purposes, and numberless articles are for one reason or another placed in the mouth, but, for no reason whatever, and all unconsciously, the fingers are with great frequency raised to the lips or the nose. Who can doubt that if the salivary glands secreted indigo the fingers would not continually be stained a deep blue, and who can doubt that if the nasal and oral secretions contain the germs of disease these germs will not be almost as constantly found upon the fingers? All successful commerce is reciprocal, and in this universal trade in human saliva the fingers not only bring foreign secretions to the mouth of their owner, but there, exchanging it for his own, distribute the latter to everything that the hand touches. This happens not once, but scores and hundreds of times, during the day's round of the individual. The cook spreads his saliva on the muffins and rolls, the waitress infects the glasses and spoons, the moistened fingers of the peddler arrange his fruit, the thumb of the milkman is in his measure, the reader moistens the pages of his book, the conductor his transfer tickets, the 'lady' the fingers of her glove. Everyone is busily engaged in this distribution of saliva, so that the end of each day finds this secretion freely distributed on the doors, window sills, furniture, and playthings in the home, the straps of trolley cars, the rails and counters and desks of shops and public buildings, and, indeed, upon everything that the hands of man touch. What avails it if the pathogens do die quickly? A fresh supply is furnished each day. Besides the moistening of the fingers with saliva and the use of the common drinking cup, the mouth is put to numberless improper uses which may result in the spread of infection. It is used to hold pins, string, pencils, paper, and money. The lips are used to moisten the pencil, to point the thread for the needle, to wet postage stamps and envelopes. Children 'swap' apples,

cake, and lollipops, while men exchange their pipes and women their hat pins. Sometimes the mother is seen 'cleansing' the face of her child with her saliva-moistened handkerchief, and perhaps the visitor is shortly after invited to kiss the little one.

"Children have no instinct of cleanliness, and their faces, hands, toys, clothing, and everything that they touch must of necessity be continually daubed with the secretions of the nose and mouth. It is well known that children between the ages of two and eight years are more susceptible to scarlet fever, diphtheria, measles, and whooping-cough than at other ages, and it may be that one reason for this is the great opportunity that is afforded by their habits at these ages for the transfer of the secretions. Infants do not, of course, mingle freely with one another, and older children do not come in close contact in their play, and they also begin to have a little idea of cleanliness."

Milk-borne Diphtheria.—The diphtheria bacillus grows well in milk without appreciably changing its flavor or appearance. Trask collected 23 diphtheria epidemics from the literature between 1895 and 1907. Fifteen of these occurred in the United States and 8 in Great Britain. The milk is usually contaminated by cases of the disease occurring on the farm or at the dairy or milk shop. In some cases the diseased person milks the cows or the same person nurses the sick and handles the milk. In two instances the outbreak was supposed to be due to disease of the cow. One of these instances studied by Dean and Todd is instructive. In certain families supplied with milk from two cows there occurred two cases of clinically typical diphtheria and three of sore throat, whereas in another family using the milk, only after sterilization, no case occurred. One of the cows had mammitis and furnished a scanty, ropy, semi-purulent, and slightly blood-tinged milk. The Klebs-Löffler bacilli were isolated in all cases and also from the milk of the cow with mammitis. Experiments justified the conclusion that the ulcers upon the udder of the cow with mammitis had become secondarily infected with *B. diphtheriæ,* probably accidentally from some apparently healthy person.

As a rule diphtheria epidemics caused by infected milk are more limited both as to numbers and area than milk-borne outbreaks of typhoid or scarlet fever.

Bacillus Carriers.—It was in the case of diphtheria that the danger of bacillus carriers was first realized. It is now known that persons who come in contact with diphtheria patients are very apt to harbor diphtheria bacilli, though they may remain in good health. It is also now well known that a certain percentage of the population at large harbor the diphtheria bacilli in their nose or throat, even though they have had no known association with the disease. Graham-Smith found that 66 per cent. of the members of the family to which

the diseased person belonged were infected; the proportion being higher (100 to 50 per cent.) in families in which no precautions were taken to isolate the sick, and much lower (10 per cent.) when such precautions were taken. Of the more distant relatives examined, 29 per cent. were found to be infected. Bacilli were found in 37 per cent. of persons in attendance on the sick. Observations of the inmates of hospital wards and institutions showed that 14 per cent. are likely to give positive cultures when diphtheria occurs among them. In infected schools 8.7 per cent. of the scholars were found to be bacillus carriers. In New York, Scholley examined 1,000 children from the tenement districts, and found 18 with virulent and 38 with non-virulent bacilli. Slack, Arms, Wade, and Blanchard took cultures at the beginning of the school year from about 4,500 pupils in the Brighton district, Boston. Diphtheria was not prevailing at the time. Nevertheless, at least 1 per cent. of all these healthy school children were found to carry morphological typical diphtheria bacilli. It is estimated that this is the average ratio in the population at large.

Ordinarily the bacilli found in diphtheria carriers under such circumstances have little or no virulence. It is possible, but not very likely, that the virulence of such strains may be raised by passing through a susceptible individual. It is probable, however, that diphtheria is kept alive in a community rather by the virulent organisms in immune persons than by these non-virulent strains. None of the children in the Brighton district above mentioned had any known association with the disease, nor did they afterward develop diphtheria. The danger of such carriers is, therefore, problematic, and, on account of their large number, it is a question whether they should be isolated. The dangerous carrier is he who harbors the virulent strain, and this is usually obtained from the patient, convalescent, or from a third person who has come in contact with the patient. From our present standpoint it seems impractical to stamp out diphtheria from a large city by cultural tests of all its inhabitants and isolation of all carriers, especially where dependence is placed upon morphological diagnosis. Some harmless bacteria have the morphological appearance of the diphtheria bacillus. On the other hand, the control of diphtheria outbreaks in institutions, camps, on shipboard, schools, and in similar places, where a number of people are crowded together, as well as the control of epidemic outbreaks in cities and towns, depends eventually upon the recognition of carriers and their isolation.

Park points out that diphtheria bacilli of like toxic power may differ in their liability to infect the mucous membrane. Virulence, therefore, has two distinct meanings when used in connection with the diphtheria bacillus. The virulence of the bacilli cannot be accurately determined from the severity of an isolated case. The most

virulent bacillus found by Park was obtained from a mild case simulating tonsillitis. In localized epidemics the average severity of the cases probably indicates roughly the virulence of the bacillus causing the infection. However, individual susceptibility and the character of the associated bacteria are important factors in determining the severity of the disease.

The length of time it requires for diphtheria bacilli to disappear from the throat and nose varies greatly. Beebe and Park found that in 304 of 605 consecutive cases the bacilli disappeared within 3 days after the disappearance of the false membrane. In 176 cases they persisted for 7 days, in 64 cases for 12 days, in 36 for 15 days, in 12 cases for 3 weeks, in 4 cases for 4 weeks, and in 2 cases for 9 weeks. In some instances the virulent organisms may remain for months. The disappearance of the bacilli from the throat and nose cannot be hastened by the usual injections of antitoxin, although Price states that diphtheria antitoxin applied locally hastens the disappearance of the bacilli. Diphtheria antitoxin, when injected subcutaneously, protects the individual but does not harm the bacilli. Careful attention to the hygiene and cleanliness of the mucous membranes may hasten their disappearance, and this is favored by copious washing of the throat and nose with large volumes of physiological salt solution. Antiseptics, such as silver nitrate, applied locally seem to be of little service.

In recent years other measures have been proposed to rid the mucous membranes of diphtheria bacilli. A serum containing agglutinins has been used with some success. This serum in powdered form is blown into the throat. The diphtheria bacilli are thereby agglutinated and may then be more readily washed away by gargling and douching. In case these procedures fail, a substance proposed by Emmerich known as "pyocyanase" may be used. This contains a ferment from bouillon cultures of the *Bacillus pyocyaneus.* It is applied locally and acts by its power of bacteriolysis.

Encouraging results have recently been reported by "over-riding" the throats of diphtheria carriers with suspensions of *Staphylococcus pyogenes aureus,* which are sprayed into the throat and nose. The method was introduced by Schiotz in 1909, who reported the prompt disappearance of diphtheria bacilli in six carriers. Page, also Catlin, Scott and Day, Lorenz and Ravenel, and others, have reported successful results.

Hewlett and Nankivell, and also Petruschky, report encouraging results in clearing up diphtheria carriers by the subcutaneous injection of a diphtheria vaccine.

We must acknowledge that all these measures often fail. The relief of bacillus carriers is one of the rewardful problems in preventive medicine.

Resistance.—The diphtheria bacillus has less resistance to adverse conditions than the majority of the spore-free bacteria. It is more readily destroyed by light, heat, and disinfecting substances than the typhoid bacillus. In this regard it corresponds more to the frailer streptococci. Under certain circumstances the diphtheria bacillus resists drying for a long time. When buried in the false membrane or other albuminous substances, they may remain virulent for some months.

Immunity.—Immunity to diphtheria is very largely an antitoxic immunity and persists for some months or years following a natural attack of the disease. Frequently immunity is of short duration, and second and third attacks are not uncommon. The fact that healthy persons may harbor virulent bacilli upon their mucous membrane for a long time without contracting the disease shows that other factors are involved. These predisposing causes are inflammations or lesions of any kind of the mucous membrane, depressed vitality due to bad air, overcrowding, poor food, etc. Persons vary markedly in susceptibility. During the first 6 months of life there is but little susceptibility. Children between the ages of 3 and 10 are most susceptible; after that age the susceptibility again decreases. It is known that guinea-pigs born of immunized mothers inherit a certain degree of resistance, which may explain the relative insusceptibility of children under 6 months. This may also be accounted for by the diminished danger of exposure of babies during this age, especially in those that are breast-fed. Mother's milk, even colostrum, contains protective antibodies, which are absorbed by the infant, and thus may protect it.

Prevention.—Control of Outbreaks in Institutions.—Diphtheria frequently appears in asylums, hospitals, jails, on shipboard, and similar places. Under these conditions of crowding the disease has a highly contagious tendency. It may, however, be controlled with every assurance of success by the application of well-tried measures. It is customary first of all to give a prophylactic dose of antitoxin to all the persons within the institution, including both inmates and administrative force. This, however, must be regarded more as a measure of temporary personal protection than as a radical means of stamping out the infection. It is not possible by the use of diphtheria antitoxin alone to wipe out diphtheria. The bacilli remain in the throats of the immunized and the disease continues to crop out after the antitoxic immunity has passed away, which may be a matter of only a few weeks. When diphtheria antitoxin is used as a prophylactic, the dose is 1,000 units, which should be repeated every ten days or two weeks—as long as the danger persists.

The most important measure to suppress diphtheria in an institution is to isolate all cases and all carriers. This is possible in an institution, although not very practical among the population at large.

The isolation of both cases and carriers is the most important and radical of our preventive measures. In the case of institutions, jails, ships, and similar places all those who show cultures containing organisms which morphologically resemble the diphtheria bacillus should be isolated, whether the strains are virulent or not.

The bacilli frequently grow in the mucous membrane of the nose and nasal pharynx without symptoms indicating their localization. Unless cultures are taken from the nose, many carriers will be overlooked, leaving a large loophole in our preventive measures. Ward and Henderson in a public school epidemic in Berkeley in 1907 found that all attempts to isolate infected children had no effect on the epidemic so long as they made throat cultures alone. When they took both nose and throat cultures and quarantined all the children showing positive cultures, the epidemic stopped.

Convalescents should not be released from quarantine until at least two cultures taken from both the nose and throat are negative.

In addition to the above-mentioned measures, care must be taken that the infection is not spread by the use of cups, spoons, dishes, towels, handkerchiefs, and other articles used in common. The infected discharges should be rendered harmless at the bedside, and all objects that come in contact with patients or carriers should be disinfected. A general disinfection with formaldehyde may be practiced, but in a well-ordered institution the usual cleanliness of floors, walls, and other surfaces will suffice.

Control of Epidemics.—The principles which guide us for the control of outbreaks among the population at large are precisely the same as those described for the control of epidemics in institutions. The only difference is that in the population at large it is more difficult, if not impossible, to apply the one real important measure, namely, that of isolating the carriers. What is needed is a convenient and reliable method of distinguishing the virulent and dangerous bacilli from those that look like diphtheria bacilli but lack pathogenic power and danger to man.

In almost all communities diphtheria is now one of the diseases which must be reported to the health authorities. The houses are placarded and the cases isolated. There is no great objection to treating a case of diphtheria in the household provided the patient and the nurse may also be quarantined from the rest of the household. Under these circumstances and with intelligent care and disinfection at the bedside there is little danger to the rest of the family; but the great menace that some of the members of the family will harbor bacilli of a dangerous type and transmit them to others makes it advisable to treat all cases of diphtheria in a special hospital.

The prompt and early diagnosis of diphtheria has now become one

of the routine measures of board of health laboratories. This example in the case of diphtheria could be extended with advantage to the other communicable diseases for which we have satisfactory laboratory aids. Especially commendable is the general practice of refusing to lift the quarantine until two successive cultures prove negative.

Disinfection in diphtheria should be applied especially to the secretions from the mouth and nose. These may be received upon a piece of gauze and burned. For the hands and other objects bichlorid of mercury (1-1,000), carbolic (2½ per cent.), formalin (10 per cent.), tricresol (1 per cent.), are efficient. As a terminal disinfectant formaldehyde gas may be used, but the ordinary fumigation, as practiced by Boards of Health, seems to have little influence in checking the spread of the disease. Evidence is accumulating that the infection usually comes from persons rather than from things. Bed linen, towels, and other fabrics should be boiled or steamed.

Personal Prophylaxis.—In individual cases diphtheria may be avoided by the use of diphtheria antitoxin. The antitoxic immunity, however, depends upon the free circulation of the antibodies in the blood, and as the antitoxin is gradually eliminated it cannot be depended upon to protect more than 2 or 3 weeks.

Diphtheria antitoxin is a specific and sovereign remedy. When given in sufficient amounts during the first 24 hours of the disease it reduces the mortality to practically nil. Ordinarily 500 units are sufficient for prophylactic purposes, but 1,000 units are preferable, as this amount produces an immunity of higher degree and longer duration. When the exposure to the infection continues the antitoxin may be administered at successive intervals of about 2 or 3 weeks. Upon the first appearance of sore throat, fever, or other suggestive symptoms in persons who are exposed to diphtheria a full dose of 3,000 to 10,000 units should be administered without delay. In order to obtain the full life-saving benefits of diphtheria antitoxin, it should be given early in the disease. Time is the most important factor. When the damage to the cells has been done, it may be too late. It is not always advisable to wait for bacterial confirmation. Personal prophylaxis is further favored by the individual having his own glass, cups, spoons, towels, etc., and exercising personal cleanliness, especially concerning the hands and all objects placed in the mouth. Physicians, nurses, and others who come in close contact with the patient should guard against droplet infection.

PREVENTION OF POST-DIPHTHERITIC PARALYSIS

It has been observed that post-diphtheritic paralysis is more frequent since the use of antitoxin than before the days of serum therapy.

This is due to the fact that many cases now recover that would formerly have died. It is also due to the fact that diphtheria antitoxin is sometimes used too late, thus neutralizing only the acute effects of the *toxin,* but not neutralizing the after-effects of the *toxon,* which acts specifically upon the nerves. The prevention of post-diphtheritic paralysis, therefore, consists in giving sufficient amounts of antitoxin early in the disease. The antitoxin does not influence the paralysis after it has once appeared.

PREVENTION OF SERUM SICKNESS

This subject may appropriately be considered here, although it is a condition that may follow the injection of any alien serum into the system. Serum sickness is a syndrome which frequently follows the injection of horse serum into man. The symptoms come on after about 8 or 10 days following the injection. They consist of various skin eruptions, usually urticarial or erythematous in character; also fever, edema, glandular enlargements, rheumatic-like pains in the joints, and albuminuria. The eruptions may be either local or general, and sometimes resemble that of scarlet fever or measles. Serum sickness has nothing to do with the antitoxin, but is caused entirely by the foreign proteins contained in the horse serum. It may be produced with normal horse serum as well as with antitoxic horse serum. The studies upon anaphylaxis have thrown much light upon the nature of this complication. The serum of some horses is much more apt to produce the syndrome than that of other horses. A serum that is several years old is perhaps less apt to produce these reactions than a fresh serum. Manufacturers of antitoxin, therefore, prefer to keep their serum in the ice chest some time before they place it upon the market, although this a doubtful expedient. The occurrence and severity of the symptoms are in direct proportion to the amount of foreign protein injected. Fortunately, this form of anaphylactic reaction soon passes away and is never serious. Under certain circumstances, however, there may be an accelerated or immediate reaction threatening in its consequence or even leading to death. Rosenau and Anderson have collected some 19 cases of sudden death following the injection of horse serum, and they know of more instances which have not appeared in the literature. This unusual and serious complication comes on within 5 or 10 minutes of the injection, and is characterized by collapse, unconsciousness, cyanosis, labored respiration, and edema. The heart continues to beat after respiration has ceased. The entire picture is an exact counterpart of the anaphylactic shock so readily reproduced by second injection of horse serum or other foreign protein in the guinea-pig. Contrary to the experimental work on the lower animals, most of the cases of

sudden death in man follow the first injection of horse serum. The serious symptoms and death in these cases are not due to any inherent poisonous property in the antitoxic serum, but result entirely from a hypersusceptibility of the individual. Just how man becomes sensitized in these cases is not known. Most of the cases, however, occur in asthmatics or in persons who gave a history of asthma or discomfort when about horses. This is a practical and important point, and should be inquired into before horse serum of any kind is injected. Horse serum should not be injected into such individuals unless the indications are clear, and then only with a statement as to the possible outcome.

In order to prevent this serious complication a small quantity may first be injected, 1 or 2 drops, and after waiting an hour the remainder may be given. Vaughan proposed 0.5 c. c. as the trial dose, but this is excessive, as some of the fatal cases have followed the injection of about 1 c. c. It is known that in man, as in the experimental cases in the guinea-pig, the severity of the symptoms bears a definite ratio to the amount of serum and the mode of injection. Thus, second injections in the guinea-pig are much more fatal when given directly into the circulation than into the subcutaneous tissue. It is sometimes advisable to give antitoxic sera directly into the circulation, but in the susceptible persons under discussion this would be hazardous.

Friedberger and Mita[1] found it possible to avoid all symptoms of anaphylaxis in experimental work with guinea-pigs by injecting the serum extremely slowly. When thus introduced animals are able to tolerate an amount far beyond the ordinary lethal dose.

Historical Note.—A complete summary and bibliography of diphtheria up to 1908 will be found in the system edited by Nuttall and Graham-Smith entitled "The Bacteriology of Diphtheria," containing articles by Löffler, Newsholme, Mallory, Graham-Smith, Dean, Park, and Bolduan; Cambridge University Press, 1908.

The original clinical description of the disease is, by common assent, attributed to Bretonneau in 1826: Traité de la diphthérite. Des inflammations spéciales du tissu muqueux et en particulier de la diphthérite ou inflammation pelliculaire, connue sous le nom de croup, d'angine maligne, d'angine gangréneuse, etc., Paris.

The bacillus of diphtheria was first cultivated and adequately described by Löffler, 1884: Untersuchungen über die Bedeutung der Mikroorganismen für die Entstehung der Diphtherie beim Menschen

[1] Friedberger, E., and Mita, S.: "To Prevent Anaphylaxis in Serotherapy" ("Methode, grössere Mengen artfremden Serums bei überempfindlichen Individuen zu injizieren"), *Deutsche med. Wochenschr.*, Berlin, Feb. 1, XXXVIII, No. 5, pp. 201-248.

bei der Taube und beim Kalbe. *Mitth. a. d. K. Gesundheitsamte,* ii, 451.

The classical article in which Behring and Kitasato announced their discovery of diphtheria antitoxin in 1890 will be found in *Deutsche med. Wochenschr.,* xvi, 1113. Ueber das Zustandekommen der Diphtherieimmunität und die Tetanusimmunität bei Tieren.

Ehrlich's important work, in which he laid the foundations of his side-chain theory and established the present satisfactory method of standardizing diphtheria antitoxin, will be found in the following: Die Werthbemessung des Diphtherieheilserums und deren theoretische Grundlagen. Klin. Jahrb., Jena, v, 6 (2), 1897, pp. 299-326. Ueber die Constitution des Diphtheriegiftes. *Deut. med. Woch.,* Leipzig, v, 24 (38), 1898, pp. 597-600. Croonian lecture. On Immunity with Special Reference to Cell Life. Proc. Roy. Soc., London, v, 66, pp. 424-448, pls. 6-7.

The official method for standardizing diphtheria antitoxin in this country and the principle upon which it is based are described by Rosenau (1905), The Immunity Unit for Standardizing Diphtheria Antitoxin (based on Ehrlich's normal serum). *Hygienic Laboratory Bull.* No. 21, P. H. and M. H. S., Washington, Govt. Print. Office, 92 pp.

MEASLES

Measles is usually taken as the type of a contagious disease because it is one of the most readily communicable of all diseases, in this regard ranking with smallpox. As a cause of death it ranks high among the acute fevers of children. Measles is an infection peculiar to man, although experimental measles has recently been produced in monkeys. The virus is contained in the blood, as has been shown by Hektoen, who thus transmitted the disease from man to man. More important from the standpoint of prevention, the virus has been demonstrated in the secretions from the nose and mouth by Anderson and Goldberger. The period of incubation is quite constant (from 9 to 11 days), and the rash appears quite uniformly on the 13th or 14th day after the infection. In Hektoen's two experimental cases the eruption appeared on the 14th day. The cause of measles is not known.

Measles is more or less constantly present in all large cities in the temperate zone; it is less common in the tropics. Measles frequently becomes epidemic, usually in the cooler months, in this respect resembling smallpox. The epidemics recur cyclically, at irregular intervals. Levy and Foster noticed that in Richmond, Va., epidemic outbreaks recurred at intervals of about 3 years. They were able to predict and warn against an epidemic prevalence of the disease in the

winter of 1910. During 1909, 40 cases of measles occurred in Richmond, but during this year the disease showed no special tendency to spread. In the middle of February, 1910, 8 cases occurred among the pupils of one school and the infection showed a high degree of communicability. According to the history of the disease, an epidemic year was due and an epidemic was predicted. Over 2,000 cases occurred with 26 deaths.

Measles is highly contagious during the preëruptive stage, when the nature of the disease is not recognized and when most of the damage is done; it remains contagious for a variable time during convalescence. Recent experimental evidence and clinical experience plainly indicate that the infection of measles soon dies out, and that there is little danger of transmitting the infection after the temperature returns to normal. An isolation of two weeks from the onset of the disease is sufficient in public health work; health officers, however, adopt arbitrary times. Thus, in Detroit cases of measles are isolated one week; in Buffalo, Concord, New York, Providence, and Yonkers, two weeks; in Brookline and Fall River, two weeks after the eruption fades; in Boston, two weeks after recovery; and three weeks in Montclair, N. J., New Bedford, Mass., Ottumwa, Iowa.

Immunity.—One attack of measles usually confers a rather definite protection against subsequent attacks; second attacks, however, are more common than in the other eruptive fevers. Some persons have the disease three or four times. As with smallpox, there appears to be no natural immunity to measles—man is exquisitely susceptible to these two infections. There appears to be a relative immunity sometimes of a high grade during the first few months of life, although measles occasionally occurs in infants of a month or six weeks.

Adults are susceptible to measles, provided they have not had a previous attack. Susceptibility to the infection does not diminish with increasing age; the disease is apparently one of childhood only on account of the chances of exposure in early life. Before the days of vaccination smallpox was also a disease mainly of childhood.

The following instances demonstrate the susceptibility of adults to measles and also the serious nature of the disease: Measles was introduced into the Faroe Islands in 1846 from Copenhagen, and over 6,000 of the 7,782 inhabitants were stricken. In 1775 it was introduced into the Sandwich Islands, and in 4 months 40,000 of the population of 150,000 died.

Measles is common in army camps, especially among troops enlisted from country districts, who are thus exposed to the infection for the first time.

Measles is often fatal both in adults and children on account of pneumonic complications. It also seems to lower the resistance to tu-

berculosis; for it is a common history to find tuberculosis develop in children following an attack of measles.

Resistance of the Virus.—In general the virus of measles is known to be much less resistant than that of scarlet fever and many other infections. The virus does not live long upon fomites. There is practically no danger of children contracting the infection from the room in which the patient was treated, even though no disinfection was practiced, provided two weeks have elapsed.

Goldberger and Anderson [1] found, as the result of experiments upon monkeys, that the virus in measles' blood is filterable; that is, may pass through a Berkefeld filter. It resists desiccation for 25½ hours, loses its infectivity after 15 minutes at 55° C., resists freezing for 25 hours, and possibly retains some infectivity after 24 hours at 15° C.

From the standpoint of our present knowledge it is evident that any of the ordinary germicidal agents sufficient to kill spore-free bacteria will serve as effective disinfectants for measles. Aside from the few scientific observations upon the viability of the virus of measles, epidemiological observations have long pointed out the fact that the virus of measles is frail and soon dies in the convalescent as well as in the environment.

Modes of Transmission.—The virus of measles is contained in the nasal and buccal secretions. While it is possible that the virus may leave the body in other secretions, it is highly probable that the discharges from the nose and mouth are the means of transmitting the infection in the vast majority of cases. We are less certain concerning the modes of entrance into the body, although it is presumed that the virus also enters by the mouth and nose; however, we lack positive information upon this point.

Mayr [2] showed in 1852 by experiments on the human subject that the buccal and nasal secretions were infective. Recently Anderson and Goldberger [3] have demonstrated by experiments upon monkeys that the nasal and buccal secretions of uncomplicated cases of measles may be at times, but are not always, infective. Hektoen [4] in 1905, as well as Goldberger and Anderson, 1911, demonstrated that the virus of measles is also contained in the circulating blood. The virus appears in the blood at least 24 hours before the eruption appears, and begins to diminish about 25 hours after the first appearance of the eruption.

It has long been assumed that the virus of measles is carried in the fine bran-like desquamating epithelium, which is one of the characteristics of the disease. Mayr long ago failed in his attempts to in-

[1] *J. A. M. A.*, Vol. LVII, No. 12, Sept. 16, 1911, p. 971.
[2] Mayr, Franz: *Zeitschr. d. k. k. Gesellsch. de Aertze zu Wien*, 1852, I, 13-14.
[3] *J. A. M. A.*, Vol. LVII, Nov. 11, 1911, p. 1612.
[4] Experimental Measles: *Jour. Infect. Dis.*, 1905, Vol. II, p. 238.

oculate children with measles by using the desquamating epithelium. Anderson and Goldberger also obtained absolutely negative results in three experiments, in which it was shown that the "scales" were not infective for monkeys. These authorities believe that it is highly probable, if not altogether certain, that the desquamating epithelium of measles in itself does not carry the virus of the disease. This conclusion is warranted by epidemiological evidence.

Measles is so readily communicable that clinicians receive the impression that the virus is "volatile." It has long been suspected that the virus is contained in the expired breath, but this is very doubtful. In fact, it may now be stated with confidence that measles is not air-borne, in the sense in which this term is usually understood. In any case, the radius of danger through the air is confined to the immediate surroundings of the patient—that is, within the danger zone of droplet infection. Droplet infection is quite possible, as the virus is contained in the secretions of the mouth and nose; furthermore, it evidently requires an exceedingly minute quantity of the virus to reproduce the disease in man, who is exquisitely susceptible to this infection.

Chapin has collected important evidence indicating that the infection of measles is not air-borne. Thus, in the Pasteur Hospital, Paris, each patient is cared for in a separate room opening into a common hall. Trained nurses exercise strict medical asepsis. In 2½ years after this hospital was opened in 1900 many cases of smallpox, diphtheria, scarlet fever, and 126 cases of measles were cared for. In no instance did measles spread within the hospital. At the Children's Hospital in Paris (Hôpital des Infants Malades), instead of being in separate rooms, the beds are separated only by partitions. Strict asepsis is observed. Of 5,017 cases there were only 7 cross-infections, 6 of measles and 1 of diphtheria. Dr. Moizard thinks that this experience proves that even measles is not air-borne, for the few cases of this disease which did arise were not all in cubicles adjoining those occupied by measles patients. Grancher in another Paris hospital had two wards in which there were no partitions, but only wire screens around the beds, simply as a reminder for the nurses. Of 6,541 patients treated from 1890-1900, 115 contracted measles, 7 scarlet fever, and 1 diphtheria. Grancher insists that measles is probably not an air-borne disease. Adjacent patients do not necessarily infect one another. At various English hospitals similar methods have beeen tried with success. These various hospital experiences indicate that the danger of aerial infection in measles is much less than is generally supposed.

The infection of measles is usually transmitted more or less directly from person to person by means of the excretions from the mouth and nose, and most often during the early stages of the disease. Measles

may be transmitted by third persons or by fomites, though such instances are rather exceptional.

Prevention.—The suppression of measles is one of the most difficult problems we have to face, for the reason that the disease is one of the most highly communicable of all infections, and for the further reason that it is most contagious during the preëruptive stage. To the student of preventive medicine the problem of measles is very similar to that of smallpox, and the final control will probably have to await a specific prophylactic measure. Improved sanitation, better hygiene, and the general advance of civilization, which have made such a marked impression upon typhus fever, relapsing fever, typhoid fever, and other "filth" diseases, have no influence whatever upon such infections as measles or smallpox.

Measles is such a common disease that parents are prone to take little pains to avoid the infection; they even sometimes purposely expose their children. This is a mistaken attitude. Special care should be exercised especially during the first five years of life, as over 90 per cent. of the fatal cases occur in this period. While it may be almost hopeless to lessen the morbidity in measles, it is quite possible to materially decrease the mortality by simply delaying the age incidence.

Clinical experience plainly indicates that few people die of measles if properly cared for. The mortality may, therefore, be decreased by careful nursing and protection, especially from pneumonia, which is one of the most dangerous complications. Newman sums up the matter of prophylaxis when he states that "the prevention and control of measles, like that of whooping-cough and tuberculosis, is largely in the hands of the public themselves."

In the present state of our knowledge the prophylaxis of measles rests almost entirely upon one measure—isolation. Chapin believes that isolation has been a failure in measles. This is because of the unrecognized but infectious preëruptive stage. "No amount of isolation after the disease is recognized can atone for the harm done before the diagnosis is made." Isolation, however, accomplishes one worthy object, viz., the prevention of further damage. Isolation, as carried out in our large cities, has had no apparent effect upon the prevalence of the disease. In Aberdeen restrictive measures apparently protected only 7 to 10 per cent. of the population.

Despite its limitations, isolation is quite worth while. Cases should be at once reported to the health officer, the house placarded, and visiting prohibited. Quarantine should not be raised nor should the child be permitted to return to school until the manifestations of the disease have disappeared. Measles may be treated in the household, but it is difficult under ordinary circumstances to prevent the spread of the dis-

ease to the other children. If the case is treated at home, the children who have not had the disease should be sent away.

Mild atypical and unrecognized cases of measles occur, but are far less numerous than such cases in scarlet fever, diphtheria, and typhoid. Clinical evidence points to the fact that "carriers" of measles are not common. The disease is usually spread directly from person to person, occasionally indirectly through a third person, or by fomites. Physicians may convey the infection to healthy children. I am convinced that I carried the disease to my own son. When measles is conveyed by a third person or by fomites it is by means of contamination with the fresh buccal, nasal, or bronchial secretions upon the hands, handkerchief, or some other object that comes in contact with the mouth or nostrils of a susceptible child. Physicians may readily avoid this danger by wearing a gown and carefully washing the hands, face, and hair, and waiting a reasonable time before visiting healthy children.

Terminal disinfection is of comparatively little value in preventing the spread of measles. After the patient is released from isolation a general disinfection with formaldehyde may be practiced, especially if healthy children are soon to occupy the playroom or bedroom. However, if from 2 to 3 weeks have elapsed, there is practically no danger in a well-ventilated, sunny, and clean room. All bedding, towels, handkerchiefs, and other fabrics that have been exposed should be boiled or otherwise disinfected.

The question of closing the schools in order to prevent the spread of measles requires consideration. If the school is closed at the beginning of an outbreak and the disease continues to spread after two weeks, little more will be gained in keeping the school closed, for it must then be evident that other factors are at work in spreading the infection. As the disease is mainly spread in the preëruptive stage, it is sufficient to examine the children each morning *before* they enter school for symptoms of a cold, infection of the eyes, running at the nose, cough, sore throat, fever, etc. All such cases should be sent home to await further developments. If these measures are taken the school may be kept open.

McVail suggests that when a child develops measles all the children exposed may be allowed to continue at school 8 or 10 days, and then excluded for a week to ten days, when those who do not develop the disease may be allowed to return. This is a rational plan used in certain districts in England. When measles breaks out in an orphan asylum, a public institution, or an encampment, the only chance of checking the spread of the disease is through the early recognition of first symptoms and isolation.

SCARLET FEVER

Scarlet fever is an acute febrile infection characterized by a diffuse eruption which appears during the first day or two of the fever, and sore throat of variable intensity. The seasonal prevalence of scarlet fever resembles that of diphtheria. The disease increases in the fall of the year, due, in part, to the gathering of children in the schools. The period of incubation is from 1 to 7 days; usually 2 to 4. In a few instances, in which individuals have been inoculated with the blood of scarlet fever patients, 3 to 4 days elapsed before the onset of symptoms. Scarlet fever is rare in the tropics; when introduced it soon dies out. There is probably always more or less scarlet fever in any thickly settled district in the temperate zone. The infection is kept alive largely through the mild and unrecognized cases. Scarlet fever varies greatly in intensity in different outbreaks. In some epidemics the death rate is 30 per cent.; in others it is practically nil.

Landsteiner, Levaditi and Prasek[1] apparently succeeded in transferring scarlet fever to chimpanzees and also to monkeys. The animals were inoculated both by applying throat swabs from scarlet fever patients to the pharynx of the animals, and also by injecting the animals with blood from scarlet fever patients. While the nature of the virus is still unknown, it seems to be present in the tonsils, tongue, blood, lymph nodes, and pericardial fluid.

The cause of scarlet fever is not known. Streptococci are almost constantly found in the throat and blood of scarlet fever cases. Klein in 1885 was the first to advocate the *Streptococcus scarlatinæ* as the specific cause of scarlet fever. Kurth assigns an etiological factor to the *"Streptococcus conglomeratus."* It is said to produce a rash in animals and men who are injected with it. The chief reasons for considering streptococci as the cause of scarlet fever are that they are constantly found in the throat of scarlet fever patients; that frequently they can be isolated from the blood of scarlet fever patients during life, and almost constantly after death; the cause of the complications and death in the majority of cases of scarlet fever is due to the streptococcus. It is probable, however, that the streptococcus plays a secondary rôle in scarlet fever as it does in smallpox; the disease itself may be due to a protozoon-like body described by Mallory, which lowers the resistance of the organism to streptococcal invasion.

Modes of Transmission.—It is taken for granted that the virus of scarlet fever is contained in the secretions from the nose, throat, and respiratory tract. The virus probably enters by the mouth and respiratory passages. Scarlet fever is not contagious during the period of

[1] *Annales de l'Inst. Pasteur,* Oct., 1911, XXV, No. 10, p. 754.

incubation; little, if any, during the period of invasion. It is most contagious during the period of eruption. Scarlet fever is readily communicable, but less so than measles or smallpox; it ranks about with diphtheria.

It has long been accepted and taught by the medical profession that the desquamation is the most infectious stage of scarlet fever, and it is now very difficult to unteach the public this erroneous view. It is now known that desquamating patients may, as a rule, be safely released from quarantine in the 6th week of their attack of scarlet fever, provided they have no mucous complications or other sequelæ. Convalescents may be a source of danger to others even after desquamation has ceased. This fact has been emphasized from a study of the so-called "return cases." Thus convalescents are released from hospital and permitted to return home; soon another case appears in one of the members of the household, who in turn comes to the hospital. Neech in a study of 15,000 cases found that the percentage of return cases was 1.86 in those cases who submitted to an average period of isolation of 49 days or under. With an average period of 50 to 56 days the percentage was 1.12; where the isolation extended to between 57 and 65 days the percentage of return cases was 1. McCullom states that in the South Department of the City Hospital, Boston, the children are kept 50 days, and no patient is released who has a discharge from the nose or an abnormal condition of the throat. Of 3,000 patients discharged from the scarlet fever ward, 1.7 per cent. of return cases occurred. McCullom is inclined to regard the infection as coming from mild and unrecognized cases of the disease rather than from the discharged case.

There is no accurate means of determining just how long a child remains infective after scarlet fever. The period of detention varies very much. Fifty days may be taken as a safe average. In New Haven and Seattle cases are dismissed after desquamation; in North Dakota 5 days after desquamation; in Ohio and South Dakota 10 days after desquamation. In various cities and states the period of isolation varies from 3 weeks to 8 weeks unless the physician certifies that desquamation has ceased. In Milwaukee, Paterson, and Pittsburg it is never maintained longer than 30 days, even if desquamation continues. Owing to our lack of knowledge on the subject, the period of isolation must remain more or less guesswork. An unduly long detention is a hardship upon the patient and the family; on the other hand, a scant period is hazardous to the community. Cases with rhinorrhea, otorrhea, throat trouble, or discharging abscesses must receive special care, as the secretions from these parts are now known to remain infective for a long time.

Many cases of walking scarlet fever present little further evidence

than a passing sore throat. These cases doubtless spread the disease, especially in schools. Third persons may carry the disease perhaps on their clothing and perhaps also as carriers. Toys, cups, spoons, thermometers, handkerchiefs, and other objects contaminated by the secretions of the mouth play the same rôle here that they do in diphtheria. Scarlet fever is not air-borne; at least the radius of infection is limited to droplet infection.

Milk-borne Scarlet Fever.—Milk is a rather frequent vehicle for scarlet fever infection. The milk is practically always contaminated from human sources. There is, however, some suspicion that streptococcal diseases of the cow may in some instances be identical with scarlet fever. This is doubtful. It is known, however, that such diseases of the udders of the cows may cause outbreaks of an infection resembling scarlet fever. Trask collected 51 scarlet fever epidemics reported as spread by milk. Twenty-five of these occurred in the United States and 26 in Great Britain. In 35 of the epidemics a case of scarlet fever was found at the producing farm, the distributing dairy, or milkshop at such a time as to have been the possible source of infection; in 3 of the outbreaks the bottles returned from infected households and refilled without previous sterilization were given as the source of infection; in 3 of the outbreaks scarlet fever persons handled the milk or milk utensils, and in 12 of the outbreaks the cows were milked by persons having scarlet fever; in one epidemic the same person nursed the sick and handled the milk; in 2 of the outbreaks the source of infection was supposed to be due to disease of the cow. A milk-borne outbreak in Washington was traced to a convalescent with a discharging ulcer on the finger. Milk-borne outbreaks of scarlet fever are sometimes very extensive.

An unusually extensive milk-borne outbreak of scarlet fever occurred in Boston during April and May, 1910. A total of 842 cases were reported from Boston and the surrounding towns of Chelsea, Winthrop, Cambridge, Somerville, Malden, and Everett. Investigation showed that most of the cases occurred on the route of a large milk contractor. Of the 409 cases in Boston, 286, or nearly 70 per cent., were on the route of this dealer; while 123, or 30 per cent., used other milk. Of the 155 cases that occurred in Cambridge, 126, or over 80 per cent., were on the route of the same dealer. About the same proportion of the cases in the other cities used the milk of this dealer. The cases appeared suddenly April 25th, and the outbreak ceased May 7th. The epidemic reached its highest mark on April 29th, when 128 cases were reported. The indications were plain that the outbreak was the result of more than a single infection. The milk was pasteurized at 60° C. for 30 minutes on April 27th, and three days following there was a notable and sharp decline in the number of cases. The source of the infection could not be traced, although it probably consisted of a

"missed" case on one of the 250 dairy farms from which the dealer obtained this particular supply of milk.

Immunity.—One attack of scarlet fever usually protects against subsequent attacks. In rare instances second attacks may occur after an interval of several years. Children under 10 are most susceptible. Sucklings are rarely attacked, though susceptible. After the 10th year the resistance to the disease increases. Ninety per cent. of the fatal cases occur in children under 10 years old. The reason why infants at the breast are less likely to take the disease may be on account of the diminished chances of the infection entering the mouth. The immunity acquired in later life may in part be due to previous unrecognized mild attacks.

Prophylaxis.—Prophylaxis in scarlet fever must necessarily be in excess of the requirements, awaiting more precise knowledge of its cause and modes of transmission. The essential features of prevention consist in isolation and disinfection. It is important to recognize the mild cases in schools through an efficient medical inspection. The answer to the question whether schools should be closed when scarlet fever breaks out varies with the circumstances. In country districts this is advisable, as the children may be kept separate, but in the cities little is gained. There is no objection to treating a case of scarlet fever in the household, provided a suitable room and trained attendant may be had. The infection may be confined to the sick room, but it is preferable to take no chances and send the susceptible individuals out of the house. The nurse should use the precautions described for diphtheria, smallpox, or measles. The physician should wear a gown and thoroughly disinfect his hands and other exposed parts after the visit. Special care must be taken with the thermometer and other instruments. The physician may find the necessary precautions and disinfection to be irksome, but they should not be shirked in justice to his other patients and the community.

The discharges from the mouth, nose, and respiratory passages, etc., should be collected upon suitable fabrics and burned. Bed and body clothing, dishes, and other exposed objects must be disinfected. Care must be taken concerning remnants of food from the sick room.

Scarlet fever is not as highly contagious as measles, but the measures employed should be practically the same until at least we have more definite knowledge concerning the channels of entrance and exit of the virus and its modes of transmission. The virus of scarlet fever is more resistant than that of measles. It clings persistently to clothing and various objects. A terminal disinfection with formaldehyde gas may be practiced, although little seems to be gained thereby. A thorough cleansing of all surfaces, with a good sunning and airing of the room, is always in order. All fabrics and other objects that have been exposed

should be disinfected. The virus is killed with agents that destroy non-spore-bearing bacteria. In Glasgow a sanitary wash-house has been established, where the clothing of scarlet fever cases may be disinfected and washed. This is a commendable example that might be followed with advantage by other cities.

Specific Prophylaxis.—Gabritschewsky first proposed the use of streptococcus vaccines as a prophylaxis against scarlet fever. He used a concentrated bouillon culture of the streptococcus isolated from a person ill with scarlet fever. The culture is killed by heating to 60° C., and 0.5 per cent. carbolic acid added. Gabritschewsky uses 0.5 c. c. of the concentrated bouillon culture in children 2 to 10 years old. For those younger half this amount, and adults twice this amount, is used. The injections are given subcutaneously in the abdomen, thigh, back, or arm. Another method of dosage is to use 0.1 c. c. for each year of the child's age with 0.25 c. c. as the minimum and 1 c. c. as the maximum. Three doses are given in periods of 7 or 10 days, the dosage increasing at each injection 1½ to 2 times the previous dose.

The only cases in which the vaccines are withheld are: (1) in those having a high temperature, although even these have received the prophylactic without evident untoward results; (2) in very young infants or patients who, from some cause or other, are greatly exhausted; and (3) in those having nephritis.

The claim is made that after 3 injections of the vaccine, and usually after 2, a complete immunity is established against scarlet fever. The immunity does not appear until 5 to 7 days after the last dose. The duration and degree of the immunity is problematical, as the vaccines have been in use so short a time. It appears that the immunity probably remains at least 1½ years.

The usual reactions, both local and general, follow these injections, but in 10-15 per cent. of the persons injected quite a different reaction occurs. Twenty-four hours after the injection there appears on the chest and abdomen, sometimes extending over the rest of the body, a punctate erythema very much like the eruption of scarlet fever, but not followed by any desquamation. The eruption lasts 1-3 days, and may be accompanied by sore throat, some swelling of the lymph glands, and often a so-called strawberry tongue. Rarely a rather severe reaction with high fever, a little albumin in the urine, and marked prostration occurs, but it rapidly disappears without permanent harm.

The reactions following the second injection are usually much less than after the first; often none at all; and after the third injection there are rarely any unpleasant features. The longer the intervals between the injections the more frequently will there be a reaction to the second and third injections. The interval of a week is considered the

most satisfactory. Richard M. Smith[1] has collected over 50,000 instances in which the killed streptococcus cultures have been injected with only 1 fatality, which was a child 2½ years old who had a severe nephritis and died on the third day after the injection.

The method has been extensively tried in Russia with favorable results, so far as one may judge from the published reports. Thus Smirnoff used the vaccines in 13 small communities in Russia where the sanitary conditions were very poor and the conditions favorable for the spread of scarlet fever. In one village there were 34 unvaccinated children, of whom 24, or 70.6 per cent., had scarlet fever; 48 vaccinated children, of whom 4, or 8.3 per cent., had scarlet fever. Of these 4, 3 came down within a week after the first inoculation, the other one 5 days after the second inoculation, too soon for immunity to have been established. The results in the other villages were equally or more satisfactory. Thus, all told, Smirnoff vaccinated 455 cases, only a part of whom allowed second injections. The results are as follows:

1 injection—	285	cases—	5	cases	of	scarlet	fever
2	"	—148	"	—2	"	"	"	"
3	"	— 22	"	—no	"	"	"	"

Of the 7 cases of scarlet fever 3 were within 7 days of first vaccination, 2 were within 7 days of second vaccination.

In the villages without vaccination 20 per cent. contracted scarlet fever and 11.1 per cent. died. In villages with vaccination 3.7 per cent. contracted scarlet fever, none died.

Yemelyanoff used the prophylactic in an epidemic in Krakow in which there were 8 or 10 new cases reported every day. Six hundred and ten persons were inoculated; of these not a single one contracted the disease. Often it was possible to keep the schools open in certain districts where inoculations were used, even though the children came from infected houses.

Equally good results are reported by a number of other Russian observers. It therefore seems that in the streptococcus vaccines we have a useful means to control epidemics of scarlet fever. Their use, with proper care, is attended with no harmful results, and they deserve a wider trial in this country.

Moser's polyvalent antistreptococcus serum has been used in the treatment of the disease, but has not been advocated as a prophylactic.

[1] *Boston Medical and Surgical Journal*, CLXII, 8, p. 242, Feb. 24, 1910.

WHOOPING-COUGH

Whooping-cough occurs in epidemics, which vary greatly in virulence, intensity, and mortality. The disease is more frequent and severe in cold climates; otherwise uninfluenced by season and weather. The cause of whooping-cough is a small bacillus, described by Bordet and Gengou.[1] This bacillus is found most readily in the beginning of the disease, in that part of the expectoration which comes from the region in which the bacteria are most active; that is, in the products from the depths of the bronchi brought up during the paroxysms. In this exudate, which is white, thick, and rich in leukocytes, the bacilli exist in considerable numbers and sometimes in almost pure culture.

Mode of Transmission.—Whooping-cough is usually transmitted directly from person to person in the same ways that diphtheria and other infections contained in the secretions of the mouth and nose are spread; it is less frequently transmitted by indirect contact or by third persons. Handkerchiefs, toys, drinking cups, roller towels, and other objects recently contaminated with the infective secretions may act as directors. It may also be transmitted by droplet infection, although in the ordinary sense whooping-cough is not air-borne.

Jahn and others called attention to the fact that domestic animals may be affected by whooping-cough, and that they may be the means of transmitting it to children. It is most frequently observed in dogs, but has also been noted in cats. Whooping-cough may be reproduced in puppies by dropping a pure culture into the nares; once started, it is readily transmitted from puppy to puppy. Klimenco[2] and Fraenkel[3] were able to produce what seemed like typical pertussis in monkeys, and Inabo[4] showed that injection of the bacillus in an ape gave rise to a typical whooping-cough with an incubation period of 13 days. Mallory and Horner[5] have shown that the bacilli are found in masses in the superficial layer of the trachea, thereby mechanically paralyzing the ciliæ.

Whooping-cough is apparently not contagious during the period of incubation, but is communicable from the appearance of the early symptoms, and is most contagious during the early stage. It may be transmitted in the late stages and after convalescence. While the virus is known to be in the secretions from the respiratory tract, all secretions from the mouth and nose must be regarded as infective.

Immunity.—There is no natural immunity to whooping-cough; all are susceptible. The greatest susceptibility is between 6 months to 5

[1] *Ann. de l'Inst. Pasteur,* Vol. XX, 1906, p. 731.
[2] *Centralbl. f. Bakteriol.,* 1908, XLVIII, 64.
[3] *München. med. Wochschr.,* 1908, LV, 1683.
[4] *Ztschr. f. Kinderh.,* June 15, 1912.
[5] *Jour. Med. Res.,* Nov., 1912, XXVII, 2, p. 115.

years. After 5 years the susceptibility decreases with age. One attack confers a definite and prolonged immunity; second attacks are rare.

Prevention.—The incubation is probably 1 to 2 weeks, but the time is indefinite, owing to vagueness of the onset of symptoms. If 16 days have passed without symptoms the danger may be considered as having passed. The long-drawn-out nature of the disease, the difficulty of diagnosis in the early stages when it is most contagious, and the fact that patients sometimes continue to spread the infection for 6 weeks after apparent recovery, make the control of whooping-cough an exceedingly difficult problem. Hence, with whooping-cough we have the same difficult problem that confronts us in the prevention of measles.

Whooping-cough should be reported, houses placarded, and the patient isolated, but the isolation in this case need not include strict confinement to a room. This, in fact, may be an unnecessary hardship to the patient, who does better out of doors. If the patient is permitted to take the air, he must avoid contact with his fellowmen and not go to school, theater, church, public assemblies, nor ride in street cars or public vehicles. Children should go out only when accompanied by an intelligent caretaker as a protection to others. It has been suggested that children with whooping-cough who are permitted their liberty should be plainly labeled with a red cross on their arm, or a yellow flag conspicuously displayed on their clothing, to serve as a warning to others.

Patients should not be released from quarantine until the spasmodic stage is over. The duration of isolation varies in different cities; thus it is 6 weeks in Montclair, N. J.; on recovery in Providence; as long as the cough lasts in Boston. In Michigan the disease is considered infectious 3 weeks before the whoop and 4 to 6 weeks after apparent recovery. The State Board of Health of that state requires disinfection of the clothing and premises before the patient is released, and forbids public funerals in deaths from whooping-cough.

Individual prophylaxis consists in avoiding the infection. The greatest care in this regard should be taken with children before the age of 5 years. Dogs, cats, and other domestic animals should be kept away from the patient, and the possibility of conveying the disease in this way must be guarded against in the susceptible.

The control of whooping-cough is a matter which is largely in the hands of the public itself. The dangerous nature of this infection should be emphasized, and people taught that it is contagious both before and after the "whoop." Mild cases which do not have the characteristic whoop spread the disease; this is especially common in adults.

Mortality.—The dangerous nature of whooping-cough is not generally realized. Thus in Glasgow the annual mortality from whooping-cough for 40 years, 1855-1894, was 13.5 per thousand inhabitants, and

exceeded that from any other acute communicable disease. In England and Wales in 1891 more deaths occurred from whooping-cough than from measles, diphtheria, scarlet fever, or typhoid fever. In our country the disease ranks high as a cause of death among children. The mortality figures would be still higher if all the deaths directly or indirectly due to it were completely reported, for the fatal termination is usually due to complications and sequelæ which occur in one-third to one-fourth of all cases. As a result of these complications the original disease is frequently lost sight of entirely in the vital statistics. According to Farr's law—that contagious diseases increase as density of population increases—the death rate from whooping-cough in our country will undoubtedly increase in our more sparsely settled states with increasing population and rapidly extending lines of railroad and other facilities, and with easy, frequent, and rapid movements of the people.

MUMPS

Mumps usually occurs between the ages of 5 to 15 years. There is decreased susceptibility both before and after this time. One attack usually confers immunity, but second attacks are by no means rare, and third attacks are sometimes reported. The disease may occur as epidemics in institutions, which usually develop slowly and last a long time. Mumps is contagious before the symptoms appear, and for some time, even 6 weeks, after symptoms have disappeared. The disease is usually spread by direct contact; rarely by indirect contact or by a third person. It is not air-borne. The virus is contained in the secretions from the mouth and perhaps the nose. The incubation is variously stated at from 4-25 days; it is usually prolonged.

Mumps is required to be reported in Maryland, Grand Rapids, and Raleigh, and placarded in Cleveland. Prevention depends upon the usual practice of isolation and disinfection.

LOBAR PNEUMONIA

Lobar pneumonia is a communicable disease which should be classified with the infectious fevers. If pneumonia were a new disease it would be regarded as "contagious," and its spread would be guarded against by isolation and the application of antiseptic principles. Many different infections are caused by the pneumococcus, but here we will consider only the specific self-limiting disease associated with massive involvements of one or more lobes of the lung, known as lobar or croupous pneumonia. The pneumococcus is found not alone in the local lung lesions, but it also invades the blood.

Pneumonia is one of the most prevalent and fatal of all acute diseases. As a cause of death it rivals and sometimes exceeds tuberculosis. According to the U. S. Census of 1890, over 9 per cent. of all deaths were due to pneumonia, and in 1900 over 10.5 per cent. Pneumonia is probably on the increase, owing to factors favoring the spread of the infection and to certain devitalizing influences of modern life which heighten susceptibility to the disease; further, more persons are now saved from the acute and fatal infections of childhood and adolescence to become victims of pneumonia later in life.

Pneumonia occurs everywhere, in all climates, at all times of the year, in both sexes, and at all ages; it is more frequent, however, during the cold months of the year. The incidence is marked at both extremes of life. It is common in children under six years; between the sixth or fifteenth year the predisposition is less marked, but for each subsequent decade it increases.

Pneumonia occurs in well-marked epidemics. Wells gives an exhaustive tabulation of the epidemics of pneumonia extending back to 1440.[1] Epidemics of pneumonia have occurred in all parts of the world: in Alaska, at Erlangen, Boston, Ireland, Italy, France, Switzerland, and on board ships. The disease has also been observed to spread in hospitals and in houses. Epidemics of pneumonia probably only occur when the organism attains an increased virulence and the factors for its dissemination are favorable. It is quite proper to regard pneumonia as pandemic.

Modes of Transmission.—The pneumococcus leaves the mouth mainly in the discharges from the mouth and nose, and enters the system through the same channels. The infection is spread directly and indirectly through the great variety of ways discussed under diphtheria and tuberculosis. Indirect transmission through cups, thermometers, handkerchiefs, and other objects contaminated with the fresh discharges occurs; and droplet infection also comes into consideration.

Resistance of the Virus.—The pneumococcus is a frail organism; it does not multiply in nature outside of the body and indirect transmission is not likely except with fresh infectious material. Even upon artificial culture media the life of the pneumococcus is brief; it must be transplanted every 2 or 3 days in order to keep it alive; it is customary in laboratories to pass it through a susceptible animal, such as a mouse or rabbit, from time to time, in order to maintain its virulence.

The pneumococcus is readily destroyed by heat; 52° C. for 10 minutes is sufficient. On the other hand, it withstands low temperatures very well. The ordinary germicidal agents destroy it quickly and with

[1] *J. A. M. A.*, Feb. 23, 1889. *Med. News*, May 20, 1905.

certainty. It may live for months in dried sputum, in which it also maintains its virulence.

Immunity.—One attack of pneumonia does not leave an immunity. In fact, one attack predisposes to subsequent attacks, as is the case with erysipelas and rheumatic fever. Man, however, must possess a certain degree of resistance to the pneumococcus, else the disease would be even more prevalent than it is, and recovery less frequent.

The mechanism of the immunity to this infection is not at all understood. Phagocytosis may play a prominent, perhaps a dominant, rôle. Protective antibodies, rather feeble, have been found in the blood serum of immunized animals, and also in the blood serum of persons who have recovered from pneumonia. The pneumococcic attack, especially the crisis, resembles an anaphylactic reaction, and, while the mechanism of immunity in this infection is probably complex, the best explanation of it at present is in terms of anaphylaxis.

Many weakening diseases diminish resistance to the pneumococcus. Pneumonia is frequent in alcoholics, and is commonly brought on by exposure to cold, to trauma, or to local irritation. It is a frequent complication of typhoid fever, influenza, Bright's disease, and other debilitating affections. Old age, as well as other enfeebling conditions, may act as a predisposing cause by lowering immunity. Other factors which predispose to pneumonia are sudden changes in temperature, irritation caused by aspiration of foreign substances, or the inhalation of dust or irritating vapors.

It should be remembered that pneumonia, like other communicable infections, frequently attacks the strong and robust.

Prevention.—The prevention of pneumonia must be based upon general principles guided by analogy from analogous infections. As long as we are ignorant of the fundamental factors concerned in the etiology and pathogenesis of the disease, our preventive measures must lack precision.

The virulent pneumococcus should not be lightly regarded as a normal inhabitant of the mouth, throat, and nose. Because the pneumococcus is very widely spread and the disease is ubiquitous, and because the associated factors which determine infection seem complicated and not well understood, are not sufficient excuses for a supine and hopeless attitude. The problem of tuberculosis has been attacked with vigor with scarcely better understanding of the fundamental problems at issue. Each case of pneumonia should be regarded as a focus for the spread of the infection. Ultimate control of the disease will probably have to await the discovery of a specific prophylactic and the recognition of dangerous carriers. Meanwhile we should think of pneumonia very much as we think of whooping-cough and influenza, as an infection which is spread from man to man through the secretions of the mouth

and nose. It is true that the pneumococcus is frequently found in the buccal secretions of healthy persons. Sternberg in 1880 first demonstrated the pneumococcus in his own saliva. Netter found it in 20 per cent. of the persons whom he examined, and the New York Commission reported its presence in from 48 to 85 per cent. Pneumococci have been isolated from the throat in 50 out of 80 normal individuals, from 66 out of 74 cases of lobar and lobular pneumonia, from 10 out of 15 "common colds," and from 14 out of 31 cases of miscellaneous diseases; in other words, many persons are pneumococcus carriers. However, there are many different strains of the pneumococcus, which vary greatly in pathogenic power. We therefore do not know how many of these pneumococcus carriers are dangerous to the host and also to his fellowmen. A somewhat analogous situation is noted in the diphtheria-like organisms in the throats of about 1 per cent. of all healthy individuals. It is probable that the virulence of the pneumococcus is higher in pneumonia than in the above-mentioned carriers, although this is a very difficult matter to determine. The findings of the Medical Commission for the Investigation of Acute Respiratory Diseases seem to make prevention a less hopeless task than at first sight appears possible from the widespread distribution of the pneumococcus. It was shown that, while a number of individuals constantly harbor virulent strains of the pneumococci in their mouths, the majority of people do so only from time to time. Individuals who come in contact with pneumonia patients are more apt to harbor the pneumococcus than those not so exposed. Patients convalescent from pneumonia may carry virulent organisms in their respiratory passages for weeks or even months.

Pneumonia should be added to the list of diseases requiring compulsory notification. Cases should be isolated at least in the same sense that tuberculosis is isolated—the discharges from the nose and throat should be burned or disinfected. If the patient is treated at home, the house should be placarded in order to discourage visiting and as an educational measure.

There is no specific prophylaxis for pneumonia. Prevention consists in avoiding the infection, sustaining the tone of the machine, care and cleanliness of the upper respiratory passages, avoiding chills, exposure, and other predisposing causes, and especially avoiding living in stuffy, ill-ventilated rooms and dusty atmospheres.

As carriers doubtless play an important rôle in disseminating this infection, the education of the public concerning certain sanitary habits should be actively continued. These include the danger of spitting promiscuously and of kissing; the proper care to be exercised in sneezing and coughing; the peril in the common drinking cup, the roller towel; and the habit of placing unnecessary things in the mouth.

It should become common knowledge that anything which tends to reduce vitality predisposes to pneumonia, such as dissipation, loss of sleep, overwork, worry, poor or insufficient food, lack of exercise, alcohol, colds, or excesses of all kinds; the atonic effect of living in overheated rooms, and the injurious effect of excessively dried and warmed air, and sleeping in warmed rooms. Cold baths, regulation of temperature and ventilation, sleeping with open windows or in the open air, are useful prophylactic measures for pneumonia as well as tuberculosis, "colds," and a large group of diseases.

Upon the Isthmus of Panama pneumonia was unduly prevalent owing to the habit of the perspiring workmen sleeping exposed to the trade winds. According to Carter, this was largely controlled by supplying the men with blankets. In Chicago, Evans believes that the prevalence of pneumonia was influenced by better ventilation of the street cars. Allaying street dust and house dust removes one of the predisposing causes of pneumonia and other respiratory infections.

Health officers may assist in the cause by disseminating knowledge concerning the disease and by enforcing antispitting regulations, by proper cleansing and oiling of streets, by requiring a stricter compliance with building and housing laws, and by the regulation of the ventilation and conditions of the air in theaters, schools, street cars, and public buildings.

INFLUENZA

The cause of influenza is assumed to be a small bacillus which is constantly associated with the disease; it was described by Pfeiffer in 1892 and 1893.[1] Influenza prevails without relation to climate, wind, weather, or telluric conditions. It occurs sporadically, in epidemics and in great pandemics. In 1889 and 1890 influenza spread to the four quarters of the globe, and, judged by the morbidity and mortality, this was the most extensive and serious pandemic that has occurred in modern times. These worldwide outbreaks usually spread from east to west.

Immunity.—Immunity to influenza is slight; in fact, one attack seems to predispose to subsequent attacks; second and third attacks are common as a result of new infections or reinfections. Influenza bacillus carriers are numerous. Males and the robust individuals in a community seem more susceptible, perhaps on account of greater exposure.

Modes of Transmission.—Influenza is spread directly from person to person. It is highly contagious in the early stages. The influenza bacillus is found in the secretions from the nose, throat, and respira-

[1] *Deutsch. med. Wochenschr.*, 2, 1892, p. 28. *Zeitschr. f. Hyg.*, XIII, 1893.

tory tract. The bacillus does not multiply outside the body and has a very feeble resistance. It grows with difficulty upon artificial culture media and soon dies out; therefore "contact" infection or the use of handkerchiefs, towels, cups, and other objects contaminated with the fresh secretions are the common modes of transmission. Influenza is kept alive in interepidemic years in carriers. Lord found the bacillus influenza in 25 to 59 per cent. of all cases with cough and expectoration in an interepidemic period in Boston.

Prophylaxis.—Prophylaxis is practically the same as for all other infections transferred by the secretions from the mouth and nose. Isolation is not always practicable, but patients for their own good as well as the protection of others should remain in bed during the febrile stage. This one measure would very largely diminish the prevalence of influenza as well as common colds. The infection could be kept out of a country by strict maritime quarantine, provided mild cases and carriers could be recognized; this, however, is not practicable. The public has not sufficient regard for influenza to tolerate aggressive measures. The disease may frequently be avoided by individual prophylaxis. During epidemics individuals should avoid theaters, mass meetings, closed and crowded cars, and close contact with their fellowmen, especially those who have catarrhal symptoms. It is quite worth while to isolate the first case of influenza in a household in order to prevent a house epidemic. This may be done on precisely parallel lines to those described for diphtheria. Influenza is especially dangerous when complicating pulmonary tuberculosis, and care should be taken to keep it out of sanitaria. Even during epidemics influenza may successfully be kept out of institutions by an intelligent quarantine. Once within the walls, it is exceedingly difficult to control. Persons who continually carry the influenza bacillus in their nose, throat, or respiratory tract should guard against exposure to wet and cold on account of the danger of reinfection. Influenza is another one of those diseases the control of which rests with the public. Education, therefore, is of prime importance. The danger from the use of the common drinking cup, the roller towel, kissing, droplet infection, handkerchiefs, pipes, toys, soda-water glasses, spoons, and other objects recently mouthed should be emphasized; spitting ordinances enforced, ventilation and overcrowding of street cars corrected, and dust allayed.

COMMON COLDS

More people probably suffer from common colds than from any other single ailment. Vital statistics give no hint of the prevalence and importance of these minor affections because the mortality is nil and the morbidity records are notoriously imperfect and difficult to collect. Could

the sum total of suffering, inconveniences, sequelæ, and economic loss resulting from common colds be obtained, it would at once promote these infections from the trivial into the rank of the serious diseases.

The common colds here considered are a group of acute infections of the mucous membranes of the nose, pharynx, tonsils, larynx, trachea, or larger bronchi. A common cold is not merely a congestion, it is an infection.

Congestion and inflammation of the mucous membrane of the upper respiratory tract frequently occur as a result of irritants other than bacteria. Thus, chemical and mechanical irritants will produce a congestion or inflammation; an increased acidity causes a flaring up of the mucous membranes, especially of the nose; and many other local and reflex causes lead to acute or chronic catarrhal conditions of these membranes, which may become exquisitely sensitive and sometimes hypersusceptible. In the absence of the proper bacteria, however, these conditions do not develop into infectious colds, and are, therefore, not communicable.

The popular fallacy of colds being due to exposure to drafts, sudden changes of temperature, and chilling of the body clings persistently in both the professional and lay mind. These are predisposing causes and will not produce a cold without the presence of the specific cause. The bacteria usually found associated with these catarrhal infections are: staphylococci, streptococci, pneumococci, influenza bacillus, the *Bacillus catarrhalis,* and other bacteria. The etiological relationship between these organisms and the disease is not always clear. Many of the above-mentioned bacteria are also found normally upon the mucous membranes of the nose, mouth, throat, and upper respiratory passages; reinfections must, therefore, be common, and predisposing factors which diminish resistance have a special importance. Common colds frequently attack the strong and robust if exposed.

Colds are contracted from other persons having colds, just as diphtheria is contracted from diphtheria. Arctic explorers exposed to all the conditions ordinarily supposed to produce colds do not suffer from these ailments until they return to civilization and become reinfected by contact with their fellowmen. A campaign to prevent the spread of the common cold would have much collateral good in aiding the suppression of tuberculosis and causing a diminution of pneumonia and other infections. Common colds occur in epidemics and have all the earmarks of a contagious disease. Colds are apt to go through all the members of a household, and outbreaks in schools, factories, and other places where people are closely associated frequently occur and result in considerable loss of time and money.

While common colds are never fatal, the complications and sequelæ are serious. These are: rheumatic fever, pneumonia, sinusitis, nephritis,

and a depressed vitality which favors other infections and hastens the progress of organic diseases.

Common colds are perhaps most contagious during the early stages. If persons would isolate themselves by remaining in bed during the first three days of a cold, they would not only benefit themselves, but would largely prevent the spread of the infection. The contagiousness and severity of colds vary greatly in different epidemics and in different seasons of the year, depending upon the particular microorganism involved and other factors not well understood.

Prevention.—The prevention of colds consists, first, in avoiding the infection, and, secondly, in guarding against the predisposing causes. Contact should be avoided with persons who have colds, especially in street cars, offices, and other poorly ventilated spaces where the risk of persons coughing or sneezing directly in one's face is imminent. Contact with the infection may further be guarded against by a careful self-education in sanitary habits and cleanliness based upon the modern conception of contact infection. Colds, like other diseases conveyed in the secretions from the nose and mouth, are often conveyed by direct and indirect contact through lack of hygienic cleanliness and a disregard of sanitary habits. Kissing, the common drinking cup, the roller towel, pipes, toys, pencils, fingers, food, and other objects contaminated with the fresh secretions will transmit the disease.

The predisposing causes of colds include a number of conditions that depress vitality and thereby diminish resistance. The mechanism by which immunity is lessened has been discussed on page 351. The principal predisposing factors in catching cold are: vitiated air, dust, drafts, sudden changes of temperature, exposure to cold and wet, overwork, loss of sleep or insufficient rest, improper food, and other conditions that lower the general vitality of the body.

A special word concerning *drafts* is necessary. Drafts in themselves cannot produce an infectious cold. The first symptom of the disease is a chill, which is not the cause, but the effect, of the infection. It is a common belief that the cold is caught when the chill occurs. The rigor frequently consists of only a transient chilliness, and it is during this time that the individual thinks he feels a draft which is producing his cold.

Drafts have no appreciable injurious effect upon persons in good physical tone. They are, however, injurious to infants, the aged, and to susceptible individuals. Drafts are particularly apt to harm persons accustomed only to still, warm air. "It is not the engine drivers and firemen of trains that catch colds, but the passengers in the stuffy carriages." Coddling renders one susceptible to drafts, partly for the reason that the vasomotor impulses which contract the blood vessels of the skin are not sent out by the nervous mechanism, and consequently

undue cooling of the part blown upon, and perhaps of the blood itself, takes place. Normally, when the wind blows upon the skin the vasomotor contraction reduces the supply of blood and the tendency to cooling is further met by a stimulus which increases heat production. While it is true that a draft can no more cause an infectious cold than it can cause diphtheria, nevertheless, it is true that a draft may be the predisposing cause by which immunity is lowered.

It is a mistake to think that the skin alone is involved in the question of drafts. The hardening of the skin as a prevention of colds is, therefore, a misnomer. The good effects of cold baths, exercise, fresh air, sunlight, and wholesome food do not consist in "hardening" the skin, but in improving the nutrition, stimulating the metabolism, helping the control of the nervous system, improving the tone of the vasomotor system, strengthening the musculature, and enriching the blood.

In preventing the ill effects of drafts, therefore, the entire organization of the body must be considered, and not the skin alone.

Other important predisposing factors to colds are mechanical defects in breathing, or the filtering power of the upper respiratory passages, also local pathological conditions, such as adenoids, polypus, deviation of the septum, chronic catarrhal conditions, all of which should receive appropriate treatment.

One of the most important predisposing factors to colds is breathing vitiated and dusty air. Good ventilation, therefore, with air not too dry nor too warm, and the allaying of dust would prevent many a cold. The bacteria producing colds are frequently found in the mouth, nose, throat and teeth of persons in good health. Cleanliness and care of these parts is, therefore, an important consideration in the prevention of common colds.

CEREBROSPINAL FEVER

Cerebrospinal fever is an infection with the meningococcus (*Diplococcus intracellularis meningitidis,* Weichselbaum). The essential lesions of the disease are chiefly focused upon the meninges of the brain and cord. The disease occurs both in localized epidemics and sporadically.

The meningococcus is a frail microorganism, closely resembling the gonococcus. Both are biscuit-shaped cocci; both grow feebly on artificial media. They are readily killed by drying, sunlight, heat, and other unfavorable conditions. They live a strict parasitic existence and cause diseases peculiar to man, with lesions which resemble each other, both as far as the character of the inflammation and the distribution of the cocci within and without the cells are concerned. As a rule, these two microorganisms are usually distinguished by the

source from which they are obtained. Otherwise the differentiation is difficult and depends upon careful cultural and biological studies.

All cases of meningitis are not caused by the meningococcus. Sporadic cases may be due to the pneumococcus, streptococcus, bacillus of influenza, the colon bacillus, the typhoid bacillus, the bacillus of bubonic plague, and of glanders. The gonococcus may also cause meningitis as a secondary complication. The epidemic form of cerebrospinal meningitis is always due to the meningococcus. Only one epidemic so far studied bacteriologically was certainly not due to the meningococcus; in this the microorganism responsible seems to have been the *Streptococcus mucosus,* or a close relative.

The first epidemic outbreak of meningitis was reported by Vieusseux in Geneva in 1805. The next year James Jackson, Thomas Welch, and J. C. Warren investigated an outbreak in Massachusetts. Since then numerous epidemics have occurred. In New York in 1904-05 there were 6,755 cases and 3,455 deaths.

The epidemiology of cerebrospinal fever differs from that of infantile paralysis in several respects. The seasonal prevalence of infantile paralysis follows the curve of the summer diarrheas (July to September), while cerebrospinal fever prevails especially in the fall and winter months. The seasonal prevalence of cerebrospinal fever is strikingly similar to that of pneumonia and influenza, and corresponds to a number of diseases, such as scarlet fever, measles, diphtheria, and smallpox, in which the principal mode of infection is believed to be through the respiratory tract, and which are supposed to be spread mainly by contact. The epidemics are usually localized. Country districts are more afflicted than cities. Children and young adults are most susceptible. Outbreaks sometimes occur in camp or on shipboard. The immunity produced by one attack is not lasting. Councilman reports five instances in which the same individual is reported to have had the disease twice.

It is probable that the meningococcus enters the system through the mucous membrane of the nasopharynx. From this position it may reach the meninges directly through the lymph channels or indirectly through the circulation. The experiments of Flexner in the monkey indicate that when the meningococcus is introduced into the cerebral cavity it escapes by a reversed lymphatic current, so that under these circumstances it may be found in the mucous membrane of the nasopharynx. Flügge, Weichselbaum, Scheurer, and others have found the meningococcus present in great numbers in the nose and pharynx in most cases of the disease during the first 12 days of illness. Park states that after the 14th day they cannot usually be found. The admirable monograph of Elser and Huntoon[1] includes a careful study of 210 cases of the disease. The most striking conclusion by these authors is the essential impor-

[1] *Journal of Medical Research,* 1909, Vol. XX, pp. 377-536.

tance of meningococcus carriers in the transmission of epidemic meningitis. The number of persons who become such carriers during an epidemic of meningitis is far greater than the number of cases of actual meningitis. Perhaps 70 per cent. of healthy persons exposed may harbor meningococci in the respiratory passages. Apart from epidemics the meningococcus can be found but rarely in healthy individuals, but apparently there are persons who, once harboring this organism in the nasopharynx, carry it permanently and thus perpetuate the disease.

Meyer, Voltmann, Furst, and Grieber[1] studied the question of carriers in cerebrospinal meningitis. They found 1.73 per cent. of meningococcus carriers in over 9,111 healthy soldiers in the Munich garrison at a time when no cerebrospinal fever was present. One examination was made from each soldier. A special study was made of 1,911 healthy persons who were examined many times, with the result that 2.46 per cent. were found to be meningococcus carriers. Of the total of 11,022 healthy persons, about 2 per cent. examined contained the meningococcus in their throats. Isolation of the carriers had no influence on the incidence of the disease, and epidemiologically they found only exceptional relationship between the carriers and the sick. In one of the years during this study numerous clinical cases occurred; in another year none, although the number of carriers remained the same both years. The authors conclude that extreme painstaking cultural detection of meningococcus carriers is unnecessary in combating the spread of cerebrospinal meningitis; that the practical benefits do not justify the care and time necessary for such work. They believe that the chief foci, aside from factors not understood in the spread of this disease, seem to be the sick and especially the mild cases. Great care should, therefore, be taken to isolate the mild case so as to diminish the number of carriers. On the other hand, in the epidemic of cerebrospinal meningitis in Texas in 1912, Thayer examined 421 persons; 59.6 per cent. were healthy carriers, as determined by the examination of stained smears. The results obtained from cultures showed 53.75 per cent. to be positive.

It is now believed that cerebrospinal meningitis is transmitted principally through the medium of healthy carriers. Only a small percentage of the carriers develop the disease. The occurrence of more than one case in families is common. In the recent Texas epidemic there were many instances in which two members developed the disease, and in a smaller number three, four, and five members became infected. The disease is undoubtedly transmitted rather directly from person to person, for the meningococcus is of such low vitality that it succumbs quickly to drying, sunlight, and other injurious influences. On account of its severity, persons suffering from the disease are decidedly

[1] *Münchener Med. Wochenschr.*, 1910, No. 30, July 26.

limited in their sphere of influence, and, as only a very small proportion of those who receive the microorganism are susceptible to it, the perpetuation and spread of meningitis must depend on the healthy carriers who pass the meningococcus on from one to another until a susceptible individual is infected and develops meningitis. The virulence of the organism is also a determining factor.

Prevention.—From our present knowledge preventive measures are clearly indicated, though very difficult to carry out. Epidemic cerebrospinal meningitis is a good example of a group of diseases in which a more precise knowledge of the modes of transmission of the disease makes it obvious that prevention is a matter of extreme practical difficulty. Flügge estimates that healthy carriers of this disease are ten times more numerous than recognized cases, and, therefore, are more than ten times as prolific a source of infection. While the isolation of the known cases will prevent a certain number of secondary cases, this measure alone cannot hope to control the disease. It is obviously impractical to undertake to make bacteriological examinations sufficient to discover all the carriers in a community of any considerable size; moreover, the control of so many carriers when discovered would require military rule. We must frankly admit that when cerebrospinal meningitis has once become epidemic it cannot be stamped out by any known means of practical application.

This does not mean that we should assume a supine attitude, for, even though the disease cannot be satisfactorily controlled, a certain number of secondary cases can be prevented. Every case and every suspected case should at once be reported to the health authorities and the patient isolated. The virus is contained especially in the discharges of the mouth and nose, and these secretions should be disinfected. The house should be placarded, visiting prohibited, and isolation practiced. These measures will help diminish the number of carriers.

Personal prophylaxis consists in avoiding the infection so far as possible, and in the use of antiseptic gargles and nasal douches. When the disease is epidemic people should keep away from large public gatherings, crowded street cars, avoid the use of public drinking cups, and the like. They should be advised to exercise more than the usual care as to personal cleanliness. The closing of the schools may, under certain circumstances, be justified. Urotropin in moderate doses has been suggested as a possible, though quite unproven, prophylactic.

While rigid quarantine is not, as a rule, effective in controlling this disease, localized outbreaks in institutions, military camps, or small towns may be kept from spreading by a strict system of isolation, even with a military cordon.

Antimeningitis serum is useful in the treatment of the disease; it is not practical as a preventive. It must be introduced into the subdural

space by lumbar puncture. The serum should be provided free of cost or at a minimum price by health authorities. Further, boards of health should provide laboratory facilities for the bacteriological diagnosis of the disease, and the recognition of carriers.

Sophian and Black [1] recommend an active immunization induced by inoculating killed cultures of the meningococcus. The cultures are grown on 2 per cent. glucose agar, and after 18 hours' growth are washed off in distilled water, shaken for 20 minutes, heated at 50° C. for 1 hour, and tested for sterility. One million bacteria are injected at the first dose, 7 days later the same number, and 7 days later 2,000,000. The injection of the dead meningococcus confers a considerable immunity, and may prove to be a valuable measure for personal prophylaxis. Chronic carriers should be inoculated with the killed cultures, and their sphere of activity should be restricted. Furthermore, they should be impressed with the danger to their fellowmen, and given careful instructions concerning spitting, sneezing, coughing; the care of fomites, such as handkerchiefs, spoons, cups, etc.; and the importance of cleanliness of the teeth, mouth, nose, and throat.

[1] *J. A. M. A.*, Aug. 17, 1912, LIX, 7, p. 527.

CHAPTER IV

INSECT-BORNE DISEASES

GENERAL CONSIDERATIONS

The fact that disease may be transmitted through the bites of insects was suspected for years, but it was not until 1893 that it was demonstrated as a new principle by Theobald Smith in the case of Texas fever of cattle and the tick.[1] Since then many diseases have been added to the list, which is constantly growing. We now know that some diseases are always transmitted through insects and others occasionally. A thorough comprehension of the subject is necessary for sanitarians and others in the fight against disease in all climates and in all places.

It may be stated as a general law that, if a period of incubation in the insect is necessary, it indicates that the parasite probably belongs to the animal kingdom and passes part of its life cycle within the insect. This constitutes the so-called extrinsic period of incubation. Malaria and yellow fever are examples of this class, which is spoken of as *biological* transmission. If, on the other hand, insects convey infection at once without a period of incubation in the insect, the transfer is a *mechanical* one; in this case the insect does not play the part of an intermediate host in the true biological sense, and there is no cycle of development of the parasite within the insect. These cases are almost all bacterial infections.

It may be stated as a general rule that the insect hosts are not harmed by the parasites which they harbor and which are pathogenic for man. Thus, the malarial protozoon is pathogenic for man, but a saprophyte for the mosquito. The same is true of yellow fever and the Stegomyia, Texas fever and the tick, plague and the flea, sleeping sickness and the tsetse fly, typhoid and the house fly, typhus fever and the louse, etc.

The *intermediate* host in the zoological sense is that animal which harbors the asexual phase of the life cycle of the parasite; the *definitive*

[1] The other names associated with the early work upon insects and their relation to disease are: Manson, Finlay, Ross, Grassi, and the U. S. Army Commission—Reed, Carroll, Lazear, and Agramonte.

host is the animal which harbors the sexual phase. Thus, in malaria man is the intermediate host, the mosquito the definitive host. In popular parlance, the insects are spoken of as the intermediate hosts in all cases.

Insects transfer infections mechanically in a variety of ways. The mouth parts, legs, or outer surfaces of the body may be smeared with the virus, which is thus simply carried to the lips, fingers or food, and thus enter the susceptible individual; or the virus may remain attached to the proboscis of a biting insect, thus transferring the infection very much as a hypodermic syringe would; or the virus may be contained in the dejecta of the insect and be scratched or rubbed into the wound made by the bite; or the virus may be contained in the digestive tube or the body cavity and be released when the insect is crushed.

Insect-borne infections are types of true endemic diseases, as they are necessarily limited in geographical distribution to the habitat of the insect host.

As a rule, only one species, or at most a single genus, acts the part of a host for any particular infection, excepting in the mechanical transference of infection by insects. Malaria is confined to Anopheles, yellow fever to Stegomyia, Texas fever to the *Margaropus annulatus,* sleeping sickness to the *Glossina palpalis,* etc. This is a question of specificity. The specific nature of some of these diseases may be due to the fact that the parasite is not pathogenic for other hosts. Thus, yellow fever and malaria cannot be given to any other animal than man, even though large amounts of the infected blood be inoculated. The disease may be specific, in the sense that it is confined to one species, because the insect conveying the infection refuses to bite other than its own host. True specificity is found in all the cases of biological transference, whereas mechanical transference of infection may take place through widely separated genera.

In some instances the virus is transmitted hereditarily through the insect from one molt to another, and even from one generation to the next. So far as known, however, hereditary transmission takes place only in those "insects" having an incomplete metamorphosis, such as the ticks. Brues suggests that the hereditary transmission of a virus is practically impossible in insects passing through complete metamorphosis, owing to the active phagocytosis during the pupal stage.

Protozoa, bacteria, and even parasitic worms may be transferred by insects. The character of the disease cannot be predicated from the nature of the insect host. Thus, ticks convey Pirosoma and also spirochetes; flies convey trypanosomes, bacteria, the eggs of worms, and a variety of other infections; mosquitoes are concerned in the transmission of the plasmodium, a protozoon, filaria, a round worm, and a filterable virus (yellow fever).

Insect-borne diseases may occur in great epidemics, as yellow fever, malaria, dengue, plague, relapsing fever, etc. When this occurs it means that the particular insect involved prevails in enormous numbers in the epidemic area.

Ticks and mites belong to the lower class of the Arachnida and are not, strictly speaking, insects (insecta), but are here considered in the same group for practical convenience.

All the parasitic animals which live upon man and the higher animals may act as go-betweens in the transportation of the microorganisms of disease. Parasites which live upon the skin are known as *ectoparasites*, in contradistinction to *endoparasites*, which live within the body. The ectoparasites may be temporary parasites, as the mosquito; or permanent, as the tick, which spends all but its earliest and last days attached to the skin of its host. Between these extremes there are parasites spending more or less of their life attached to the host; thus, the bedbug and flea are temporary, whereas lice are permanent parasites.

FIG. 17.—A SOUTH AFRICAN BLOOD-SUCKING FLY (PANGONIA), ILLUSTRATING LONG PROBOSCIS TO PIERCE HEAVY FUR OF CERTAIN ANIMALS. (Brues.)

Many of the insect-borne diseases were formerly known as "place diseases." Thus, in yellow fever it was realized that the infection was not conveyed directly from man to man, but it was believed that the house or place became infected, and it was thought that the virus lived in the soil, upon the bedding, or on the clothing. This led to the notion that *fomites* or inanimate objects played an important rôle in the transference of disease. The early studies in bacteriology gave countenance to this view until our knowledge of the part played by insects and the importance of "contacts" has placed fomites in a subordinate and oftentimes negligible position.

The prevention of the class of infections belonging to the insect-borne diseases depends upon a knowledge and thorough comprehension of three factors: (1) the disease, (2) the parasite, and (3) the insect. The suppression or control of the insect depends upon a thorough knowledge of its biology. Entomology, therefore, has become a vitally important subject so far as preventive medicine is concerned. Without an acquaintance with the life history and habits of the insect host there

will be economic loss, wasted energy, and disappointing results. The malaria mosquito is active at night and breeds in the swamps; the yellow fever mosquito is active by day and breeds about houses. Other mosquitoes have their own particular breeding and hiding places. The suppression of lice depends largely upon bodily cleanliness, the suppression of the bedbug upon house cleanliness, the dangerous fleas come largely from association with other animals, the flies from manure and decomposing organic filth, the ticks from other animals and from the infested ground and woods.

For the control of the insect-borne diseases it is not always necessary to exterminate the particular insect host. In fact, the extermination of a particular species, much more a genus, is practically a biologic impossibility. A material reduction in the numbers of the insects in a particular area will often result in an elimination of the disease.

The geographical distribution of the disease is always more limited than the geographic distribution of the insect host. Anopheles exist in many places where there is little or no malaria. Stegomyia mosquitoes are numerous in the Philippines, but the infection has not yet been carried there.

In the migration of insect-borne diseases it is usually the human host and not the insect that acts as the traveler. Insects, as a rule, do not go great distances of their own volition, and never over seas or from one country to another, unless taken in the conveyances of man or upon some higher animal. When yellow fever or malaria go from one country to another, the infection is translated in man. The infected mosquitoes are rarely transported, except occasionally upon wooden sailing vessels with water barrels that afford breeding places.

An apparent exception to this statement is the case of plague. It is the rat rather than man that spreads plague from land to land. In this case, however, the disease is primarily an infection of the rat, which carries the flea along and man is secondarily attacked. Another exception is the house and stable fly, which are known to travel a mile or more upon the wing.

An effective campaign against mosquitoes, flies, or other insect pests requires the expenditure of time and money. Further, it requires the assistance of the entomologist, the engineer, and the practical administrator. When the campaign involves extensive drainage or filling-in operations, this calls for the services of an engineer who has specialized along these lines. To attack the problem without a complete knowledge obtained from a careful study of the habits and breeding places of the particular species of insect will probably result in economic waste. Thus, in New Orleans, during the yellow fever campaign of 1905, much time and effort was saved by knowledge of the fact that the Stegomyia mosquitoes did not breed in the street gut-

ters of New Orleans. The habits and habitat of some species may vary in different localities, and a careful study of the local conditions is important to insure success. In the organization of a mosquito campaign the several branches of the work may be allotted to special divisions, each consisting of a foreman and crew. These men become skilled in their particular duties, and efficiency is thereby greatly promoted. One division should have charge of the oiling, another of the fumigation, another should seek to destroy the natural breeding places, another should attend to the screening, etc. In fly suppression one division should look after the storing and handling of horse manure, another to garbage and organic refuse, and so on. All the work must be centralized under the direction of one person with executive ability and a thorough understanding of the problem.

The suppression of insects and household vermin is essentially a question of cleanliness. The most effective measures are those which strike at the breeding places, and these will be considered in detail under mosquitoes, flies, ticks, lice, fleas, and bedbugs. Next to the suppression of their breeding places, the most important measure in a household is to starve out these pests. Food must be so protected that insects, mice, and rats cannot gain access to it. Floors and other surfaces must be kept clean, so that they do not have the least film of organic dirt upon which insects feed. There should be no cracks or crevices to collect dust and dirt, which offer comfort for insect life. Cleanliness and incessant care must not only be exercised in the household itself, particularly the kitchen, pantry, dining room, cellar, attic, and bathroom, but must also include the back yard and surroundings of the house. Old cans and broken bottles, rubbish, garbage, and general untidiness around the household afford breeding places, hiding places, or food for vermin.

All the blood-sucking parasites must be regarded as dangerous. If they do not play the rôle of an intermediate host in the biological sense, they may occasionally transfer infections in a mechanical way, or the little wounds may allow the entrance of such infections as erysipelas, the pus cocci, anthrax, tetanus, and other microorganisms. Further, all blood-sucking parasites are potentially dangerous, in that new diseases may be established as the old ones must have been established at one time through the triple alliance of host, insect, and parasite.

Science has demonstrated the danger from insects. Experience long ago decided that a healthy home must be free of insects and vermin of all kinds—it remains for the future to extend this kind of cleanliness to municipal housekeeping and rural sanitation.

The principal insect-borne diseases, their causes, and the insect responsible in each case are stated in the following table:

DISEASE	CAUSE	INSECT
	MOSQUITOES	
MALARIA (Laveran, 1880, the parasite) (Ronald Ross, 1895–8, relation to the mosquito)	*Plasmodium malariæ* (Laveran) *Plasmodium vivax* (Grassi & Feletti) *Plasmodium falciparum* (Welch) *Plasmodium immaculatum* (Grassi)	*Anopheles*
YELLOW FEVER (Reed, Carroll, Lazear and Agramonte, 1900–2)	A filterable virus	*Stegomyia calopus*
FILARIASIS (Demarquay, 1863) (Manson—also James)	*Filaria bancrofti*	*Culex fatigans, Anopheles nigerrimus* and others
DENGUE (Graham, 1903) (Ashburn and Craig, 1907)	A filterable virus	*Culex fatigans*
DISTOMIASIS-BILHARZIOSIS (Bilharz, 1851) (Katsurada, 1904)	*Schistosoma hæmatobium* *Schistosoma japonicum*	*Anopheles maculipennis* (?)
	FLIES	
NAGANA (Bruce, 1894)	*Trypanosoma brucei*	Tsetse fly (a biting fly)—*Glossina morsitans*
SLEEPING SICKNESS (Dutton, 1901, and Todd)	*Trypanosoma gambiense*	Tsetse fly—*Glossina palpalis*
PAPPATACI FEVER—3-day fever (Adriatic) (Doerr, 1909)		*Phlebotomus pappatasii*—a dipterous biting gnat
"PINK EYE"		A little fly or midge belonging to the genus *Hippelates*
PURULENT OPHTHALMIA OF EGYPT, etc.		Flies, *et al.*
POLIOMYELITIS (Rosenau and Brues, 1912)	A filterable virus coccal forms (Flexner and Noguchi, 1913)	*Stomoxys calcitrans*—The stable fly
TYPHOID, CHOLERA, DYSENTERY, etc. Contagious ophthalmia, erysipelas, anthrax, glanders and other skin infections, smallpox and other exanthems, etc.		*Musca domestica* and other flies (Mechanical transmission)
	TICKS	
TEXAS FEVER (of cattle) (Th. Smith & Kilborne, 1893)	*Pyrosoma bigeminum*, now *Babesia bigemina*	*Margaropus annulatus*
ROCKY MOUNTAIN SPOTTED FEVER (Ricketts, 1906)		*Dermacentor occidentalis* (now *venustus*)
AFRICAN TICK FEVER (Dutton, 1905)	*Spirochæta duttoni*	*Ornithodoros savignyi*
RELAPSING FEVER (Obermeier, 1875) (Ph. Ross and Milne, 1904)	*Spirochæta obermeieri*	*Ornithodoros moubata* or *Argas persicus*
PIROPLASMA CANIS	*Piroplasma canis*	
LA SPIRILLOSE DES POULES (MARCHOUX & SALEMBENI, 1903) (Ticks are not true *Insecta*.)	*Spirochæta gallinarum*	*Argas miniatus*
	BEDBUG	
RELAPSING FEVER (Obermeier, 1873)	*Spirochæta obermeieri*	*Cimex lectularius, Ornithodoros moubata, Argas persicus* and perhaps other biting insects, as fleas and lice
KALA-AZAR	*Trypanosoma leishmanii*	*Cimex lectularius* *Cimex rotundatus*

DISEASE	CAUSE	INSECT
	FLEA	
PLAGUE	*Bacillus pestis*	*Loemopsylla cheopis* and other fleas
	LOUSE	
TYPHOID FEVER (Nicolle, 1909) (Ricketts & Wilder, 1910) (Anderson & Goldberger, 1910)	(?)	*Pediculus vestimenti* Also, *Pediculus capilis*

A number of other diseases are suspected; thus, barbiero fever (*Conorhinus megistus*); pellagra (*Simulium*); hookworm (*Musca domestica*), etc.

INSECTICIDES

Practically all the germicidal agents are also insecticides. There are some exceptions to this statement, notably formaldehyde, which is one of our most potent germicides, but has little or no effect upon insect life in its gaseous state.

The action of insecticides may be considered under three classes: (1) those that act as general protoplasmic poisons, such as strong acids or alkalies, hydrocyanic acid, sulphur dioxid, etc.; (2) those that suffocate the insects, such as oily substances, and (3) those that act upon the nervous structures, such as chloroform, ether, and other general anesthetics.

Another classification considers insecticides under four groups: (1) those used by contact in liquid form or in solution; (2) those used by contact in dry or powdered form; (3) those used by contact in vapor form; (4) those used by mixing with food and which are poisonous when ingested. Insects differ markedly in their power of resisting insecticides. Those with well-developed chitinous protection, such as bedbugs and roaches, are more difficult to kill than flies, fleas, and mosquitoes.

The most practical of the insecticides for the destruction of the winged insects in an enclosed space are those that may be used in the gaseous state. Of these, sulphur dioxid, hydrocyanic acid gas, carbon bisulphid, or carbon tetrachlorid are most commonly employed and are most reliable. The uses and limitations of these and other insecticidal agents will now be considered in detail.

Preparation of the Room for Fumigation.—It is more important to tightly seal a room in which insects are to be destroyed than where only a germicidal action of the gas is looked for. Insects may escape through minute openings, and they may hide in nooks and corners

where the gas permeates slowly and feebly, or may take cover under the folds of crumpled paper or folded fabrics, and thus escape the insecticidal action of the gas. Self-preservation tempts mosquitoes and other insects as well as rats and mice to seek the light when in the presence of an irritating gas. It is, therefore, convenient to darken the place to be treated, leaving one source of light. The dead vermin may then be readily collected about this place.

Strips of paper should be pasted over doors and windows. Cracks and crevices may be caulked with towels, waste, or other suitable substance. Ventilators, fireplaces, hot-air registers, and all openings into the room must be covered, otherwise both the gas and the insects will escape. Closets and small doors should be opened and all the drawers, lockers, and similar places exposed in such a way that the gas may have free access to remote corners. Furniture should be moved away from the walls. Fabrics, paintings, instruments, bright metal work, or other objects liable to injury may be removed or covered, especially when sulphur is used.

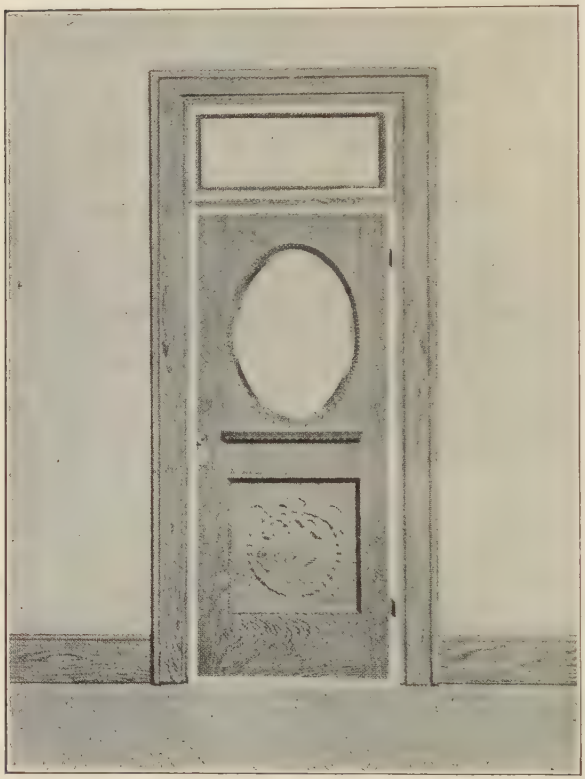

FIG. 18.—EXAMPLE OF SEALING DOORS FOR PURPOSE OF FUMIGATION.

The Relative Efficiency of Insecticides.—McClintock, Hamilton, and Lowe[1] have tested a number of insecticidal substances, the values of which are shown in Table 4, which gives a list of the substances tested and the species of insects used in the experiments, together with the quantity of each substance which, when properly transformed into vapors, was sufficient to kill the species indicated. The coefficient col-

[1] *Jour. Am. Pub. Health Assn.*, Vol. II, No. 4, Apr., 1911, p. 227.

umn shows the inverse ratio between this quantity and 8 grams, the weight of sulphur which, when burned, kills the bedbug in the 800,000 c. c. of inclosed space.

The efficient dilution of the vapors of any substance may be obtained from this coefficient by multiplying by 100,000.

For example, if one wishes to use carbon disulphid, by consulting No. 28 in the table it is shown that 24 grams were required to kill bedbugs, while only 8 grams were required of sulphur. It is therefore only one-third as strong and its coefficient is 0.3+. Its efficient dilution is 33,000.

TABLE 4

INSECTICIDES

Time of exposure—Varied as conditions required.
Column 1—Quantity used to kill the specified insect.
Column 2—Coefficient of efficiency compared with the efficiency of sulphur dioxid on bedbugs

Substance	Bedbug		Cockroach		Housefly		Clothes Moth		Mosquito	
	1	2	1	2	1	2	1	2	1	2
1 Sulphur Dioxid as Sulphur	8	1	4	2	3.2	2.5	2.6	3	3.2	2.5
2 Pyridin	8	1	4	2	2	4	1.6	5	1.6	5
3 Pyridin Bases (Merck)	5	1.6	4	2	1.6	5	1.6	5	.....	.....
4 Quinolin	8	1	8	1	.....	.....	2	4	.....	.....
5 Creosote Oil	4+	2	4+	2	2	4	1	8	8	10
6 Carbolic Acid	8	1	8	1	8	1	8	1	4	2
7 Naphthalene	8+	1	8	1	2	4	4	2	1	8
8 Kerosene	16+	0.5	16+	0.5	4+	2	4	2	4+	2
9 Anilin Oil	6.3+	1.3	6.3+	1.3	6.3	1.3	6.3	1.3	4	2
10 Cedar Oil	11.5+	0.7	11.5	0.7	8	1	2	4	1	8
11 Citronella Oil	4+	2	4+	2	2	4	4	2	1	8
12 Cloves Oil	4+	2	4+	2	2	4	2	4	1	8
13 Peppermint Oil	4+	2	4+	2	4	2	4+	2	2	4
14 Pennyroyal Oil	8+	1	8+	1	4	2	4	2	1	8
15 Australene	8+	1	8+	1	3.2	2.5	8	1	2	4
16 Turpentine (Oregon Fir)	36+	0.2	36+	0.2	36+	0.2	16+	0.5	8	1
17 Oil Pinus Palustris	16+	0.5	16+	0.5	4	2	4	2	2	4
18 Oil Turpentine	20+	0.4	20+	0.4	20	0.4	20	0.4	10	0.8
19 Turpentine (Mich. Wood)	16+	0.5	24+	0.3	16	0.5	16	0.5	.....	.....
20 Benzaldehyde	4+	2	4+	2	2	4	2	4	1	8
21 Nitrobenzol	8+	1	8	1	1.6	5	1.6	5	1	8
22 Ammonia 28%	36+	0.2	36+	0.2	20+	0.4	36+	0.2	20	0.4
23 Alcohol, Ethyl	80+	0.1	80+	0.1	80+	0.1	80+	0.1	80	0.1
24 Alcohol, Methyl	80+	0.1	80+	0.1	80+	0.1	80+	0.1	80+	0.1
25 Acetone	40+	0.2	40+	0.2	40+	0.2	40+	0.2	14+	0.2
26 Chloroform	40+	0.2	40+	0.2	16+	0.5	16+	0.5	16+	0.5
27 Ether (Ethyl Oxide)	.....	.....	.....	.....	15+	0.5	.....	.....	.....	.....
28 Carbon Disulphid	24	0.3	36	0.2	4	2	2	4	4	20
29 Carbon Tetrachlorid	40	0.2	40+	0.2	40+	0.2	40+	0.2	40	0.2
30 Chloretone	4+	2	4+	2	4	2	4	2	1	8
31 Camphor	8+	1	8	1	4	2	4	2	2	4
32*Nicotin, 80% Sol	25	4	25	4	6	20	25	40	1	100
33 Hydrocyanic Acid, as Potassium Cyanid	6.3	1.3	6.3	1.3	2	4	1	8	2	40
34 Paraform	8+	1	8+	1	4	2	8	1	1	8
35‡Formaldehyde 40% Sol	54+	0.1	54+	0.1	16+	0.5	16+	0.5	8+	1
36 Stramonium Leaves	10	0.8	10	0.8	10+	0.8	10+	0.8	4	2
37 Sabadilla Seeds	8+	.....	8+	.....	16	0.5	16+	0.5	4	2
38 Chrysanthemum Flowers	80+	0.1	80+	0.1	2.6	3	4	2	1	8

The + sign after a number indicates that this quantity was the largest used and that it was insufficient.

* Coefficient of nicotin based on 100% alkaloid.

‡ Quantity of formaldehyde to be an efficient germicide is 13½ c. c. or a coefficient of 0.625.

The best methods of generating gases for fumigating purposes are considered below. For further information concerning these substances, with special reference to their germicidal action, see Section XII.

To insure success the gas used to fumigate a room should be liberated in a large volume in a short time. If the gas is evolved slowly much of it will be lost before the room can become charged with a sufficient amount to kill the insects.

The amount of gas and the time of exposure stated in each case are the minimum. When large, leaky, or irregularly shaped spaces are to be fumigated, the amount of gas should be increased and the time of exposure prolonged. It is also advisable to generate the fumes in as many different places as practicable, as this favors rapid diffusion.

Sulphur.—Sulphur is one of the most valuable insecticides we possess. It may be used either as a gas—SO_2—or in its powdered form—flowers of sulphur.

Sulphur dioxid is destructive to all forms of life. It will kill mosquitoes, flies, fleas, roaches, bedbugs, and all kinds of vermin, including rats and mice. While sulphur dioxid is one of the most dependable insecticides it is a rather feeble germicide. It is limited in practice on account of its destructive and corrosive action. This destructive action results from the sulphurous acid and sulphuric acid produced in the presence of moisture. Fortunately the dry gas is quite as poisonous to mosquitoes, flies, rats, mice, etc., as the moist gas. Dry sulphur dioxid, however, has absolutely no germicidal value. Dry sulphur dioxid does not tarnish metals, does not rot fabrics, and does not bleach pigments. Fumigation with SO_2 may, therefore, be done with little damage to property on dry days. Metal work, fabrics, and pigments that cannot be removed from the room may be protected from the sulphur fumes by simple mechanical devices.

Sulphur dioxid may be produced either by burning sulphur or by liberating liquefied sulphur dioxid. The methods of generating the gas will be found on page 997. One pound of sulphur burned for each thousand cubic feet of air space or two pounds of liquefied sulphur dioxid and an exposure of two hours is sufficient to kill mosquitoes, flies, and other insects in a small tight space. Three to four hours are ample for rats and mice. If the space is large or leaky the amount of gas should be increased and the time of exposure prolonged. Sulphur dioxid has surprising power of penetration through clothing and fabrics. In very dilute proportions it will in one hour's time kill mosquitoes even when hidden in eight layers of toweling. It has absolutely no power of penetration when used as a germicide. This substance, which has so long been disparaged as a disinfectant because it fails to kill spores and many spore-free bacteria under certain condi-

tions, must now be considered as holding first rank as an insecticide. For consideration of sulphur dioxid as a germicide see page 997.

Flowers of Sulphur.—Sulphur in its dry, powdered state is useful against a number of parasites. In this form, however, it has little use as an insecticide in preventive medicine, not being efficacious against bedbugs, ants, roaches, or fleas.

It may be applied in several ways, the simplest of which is to sprinkle the dry sulphur about the places where insects are found. Flowers of sulphur may also be combined advantageously with other insecticides, such as kerosene emulsion, resin wash, or soap wash. It should first be mixed into a paste and then added to the spray tank in the proportion of about 1 or 2 pounds to 50 gallons. It is particularly efficacious for the destruction of the mites and rust of plants and fruits.

Sulphur in the form of an ointment is particularly obnoxious to ticks and other ectoparasites. The itch-mite (*Sarcoptes scabiei*) is very susceptible to the flowers of sulphur, which is, therefore, one of the ingredients of almost all ointments used in this skin affection.

Sulphur dips are used to destroy the mites on domestic animals. These dips ordinarily contain 1 part of lime to 3 parts of sulphur or tobacco. It is common experience that, while these sulphur dips may be depended upon to destroy the mites, they do not destroy the eggs, hence the treatment should be repeated in about 10 days, which permits time for the eggs to hatch and develop into adults.

Formaldehyde.—Formaldehyde, while holding the front rank as a germicide, is a feeble insecticide. The gas seems to have no effect whatever upon roaches, bedbugs, and insects of this class even after prolonged exposure to very high percentages. As a differential poison formaldehyde gas is a very remarkable substance. It destroys bacteria almost instantly, but, while it is irritating to the higher forms of animal life, it is not very toxic. I have repeatedly found that roaches and other insects with strong chitinous protection seem unharmed after 12 hours' exposure to formaldehyde gas in very strong atmospheres of the gas, in air-tight disinfecting chambers. Mosquitoes may live in a weak atmosphere of the gas over night. It will kill them, however, if the gas is brought in direct contact with them in the strength and time prescribed for bacterial disinfection.

When a weak insecticidal gas is used it is much more difficult to obtain direct contact between the gas and the insects than between the gas and germs, because the sense of self-preservation aids the former in escaping from the effects of the irritating substance. Mosquitoes and other insects hide in the folds of towels, bed clothing, hangings, fabrics, and out-of-the-way places where the formaldehyde gas does not permeate in sufficient strength to kill them. The gas is

polymerized and deposited as paraform on the surface of fabrics which prevent its penetration, and large quantities are lost by being absorbed by the organic matter of woolen fabrics. Mosquitoes have a lively instinct in finding cracks or chinks where fresh air may enter a room or other places where the gas is so diluted that they escape destruction. Therefore, formaldehyde gas, as well as other culicides, cannot be trusted to kill all the mosquitoes in a room which cannot be tightly sealed. On account of its feeble action, formaldehyde is not recommended as reliable.

For the best methods of evolving formaldehyde gas, the quantities to be used, and other details of the process, see page 993.

Formaldehyde gas in watery solution, known as formalin, is useful for the destruction of flies. Small quantities of dilute formalin (4 per cent.) placed in saucers about the room attract flies. They drink the fluid, which soon kills them.

Pyrethrum.—Pyrethrum is a popular and much used insecticide because it is comparatively cheap and non-poisonous to man and the higher animals. It is also non-corrosive, but unfortunately it is not very powerful for the destruction of roaches, ants, bedbugs, flies, fleas, mosquitoes, etc. It has no germicidal action.

Pyrethrum, also sold under the names of Buhach or Persian insect powder, or simply "insect powder," is the flowers of the *Chrysanthemum roseum* and the *Chrysanthemum carneum,* both hardy perennials and resembling camomile in appearance. According to Kalbrunner, 4 grains of the pure powder sprinkled on a fly in a vial should stupefy it in one minute, and kill it in 2 or 3 minutes. It acts on insects externally through their breathing pores. When brought in direct contact with them it is fatal to many forms of biting and sucking insects, such as roaches, flies, and ants. It may be used either as a dry powder or by its burning fumes. As a dry powder it may be used pure or mixed with flour, in which form it should be puffed about the room, especially into cracks.

When pyrethrum powder is ignited it smolders, giving off fumes which stun, but do not always kill, mosquitoes.[1] It is not, therefore, a dependable insecticide. This uncertainty and the price of pyrethrum restrict its field of usefulness.

Pyrethrum fumes do not corrode metals or act injuriously upon fabrics and pigments. However, a slight brown deposit is occasionally left on exposed surfaces which may stain linen a yellowish color. This deposit or stain is readily washed out, or soon fades.

Pyrethrum powder has been used very much in those cases where sulphur is prohibited on account of the danger of damage to paintings,

[1] Tobacco smoke and other substances which produce dense fumes, particularly those containing pyroligneous products, will kill mosquitoes.

fabrics, tapestries, metal work, musical instruments, upholstered furniture, and the like. It is used in the proportion of 2 pounds per 1,000 cubic feet of air space, the exposure being for not less than 4 hours. As its insecticidal effect is uncertain, it is necessary carefully to sweep up and burn all the mosquitoes that have been stunned and are apparently dead after the fumigation. Most of these mosquitoes will be found on the window sill or on the floor close to the window, where they are attracted by the light in their efforts to find an exit to escape the fumes. Advantage should be taken of this tendency of the mosquito to seek the light by darkening all but one window.

Sheets of paper containing some sticky preparation may be placed upon the floor and upon the window sill in order to catch the mosquitoes. A satisfactory adhesive preparation may be made by dissolving, by the aid of heat, 65 parts of colophony resin in 35 parts of castor oil. This simplifies the collection and disposal of the insects.

Pyrethrum powder should be distributed in pots or pans and set on fire with a little alcohol, which should first be sprinkled over it. The quantity apportioned to any one pot or pan should not exceed 1½ inches in depth, if the exposure is to be for 4 hours. The pots and pans should be set on bricks to prevent scorching the floor.

Much of the pyrethrum upon the market is impure, which further weakens what is a feeble insecticide at best.

Phenol-camphor (*Mim's Culicide*).—Camphophenique or phenol-camphor is prepared by rubbing up equal weights of phenol crystals and camphor. It may be more conveniently prepared by first liquefying the phenol by gentle heat and then pouring it over the camphor. The camphor and phenol combine to form a new chemical compound, which remains fluid at ordinary temperatures. This preparation was first used on a considerable scale during the yellow fever epidemic in New Orleans toward the close of 1905 at the suggestion of Mr. Mim, the city chemist. At this time I took the opportunity of making a number of tests with Dr. Metz concerning the culicidal value of this substance. The effect of the fumes on mosquitoes was later studied by Berry and Francis. When phenol-camphor is moderately heated it gives off dense fumes, which rise rapidly and diffuse slowly, and after 30 to 60 minutes, depending upon the amount employed and the temperature of the air, the fumes condense and are deposited as a slight moisture on all exposed surfaces. As a culicide phenol-camphor may be compared to pyrethrum; the fumes stun the mosquitoes, but do not invariably kill them. The fumes are somewhat irritating to the mucous membranes, especially the eyes; they may cause dizziness, headache, cloudy urine, and other mild symptoms of phenol poisoning in susceptible individuals much exposed to their inhalation. The fumes of phenol-camphor do not tarnish metals, rot fabrics, or bleach pigments. They, however,

have the disagreeable property of softening the varnish of surfaces on which they condense. On account of its slight power of diffusion, relatively high cost, and uncertainty of action, it cannot take the place of sulphur except in the parlor, pilot house, and other compartments where sulphur is prohibited on account of the damage it produces. Compared with pyrethrum, phenol-camphor is less expensive, more certain, and not so objectionable to the housekeeper. Its use involves a little more care and intelligence than that required for the simple burning of pyrethrum. If it is overheated it will take fire, and no culicidal action is produced. Goldberger concludes that, for use on a large scale, as in times of epidemics, in the hands of trained fumigators, phenol-camphor is, on the whole, to be preferred to pyrethrum, being more easily transportable on account of the small bulk required, and because the fumes condense quickly and the room may, if desired, be entered in an hour and the apparatus removed, thus making it possible to fumigate a larger number of rooms in a given time with less labor than in the case of either sulphur or pyrethrum.

Phenol-camphor is used in the proportion of 4 ounces to every thousand cubic feet of air space, and with an exposure of 2 hours. In this proportion and time the film of condensation is slight and is rapidly dissipated after the doors and windows are opened. The preparation of the room is the same as that described above. The phenol-camphor apportioned to the room to be fumigated should be distributed in agate-ware basins, not more than 8 to 10 ounces to any one basin. Each basin is set over an alcohol lamp at such an elevation and in such a manner as will permit a rapid evolution of the fumes. Care must be taken not to heat the basin so quickly as to cause the liquid to become overheated and take fire. This point must first be determined experimentally for each type of lamp used. One of the small brass alcohol vapor lamps to be found on the market serves excellently. As a safeguard against accidents the lamp should stand in a pan containing about one-half inch of water. The basin containing the phenol-camphor may be set upon a section of galvanized iron stove-pipe, at one end of which sectors are cut out so as to form legs of a length equal to the height of the lamp; just below the upper margin of the pipe a series of holes are punched so as to provide for draft. The stove-pipe should be of such a length as to support the basin containing the phenol-camphor about 10 inches above the flame. This ingenious and simple device, suggested by Berry and Francis, acts as a chimney, protects the flame, is relatively cheap, and has proven satisfactory.

Hydrocyanic Acid Gas.—Hydrocyanic acid gas is extremely poisonous to all forms of life. It kills roaches, bedbugs, mosquitoes, fleas, flies, rats, mice, and other vermin with great certainty and very quickly. It is much less poisonous to the higher forms of vegetable life, al-

though it has a certain amount of germicidal power. Hydrocyanic acid gas is much used in greenhouses for the destruction of insect pests and for scale and other parasites of fruit trees. The gas has a distinct place in the disinfection of granaries, stables, ships, barns, outhouses, railroad cars, and other uninhabited structures infested with vermin. It is also extensively used in flouring mills against weevils, in railroad cars against bedbugs, and in tobacco warehouses against insects in general. It should be used in the household only with the greatest precaution, as the least carelessness with it would probably mean the loss of human life. It has the marked advantage that it does not harm metals, fabrics, or pigments, and may be used in the most expensive drawing rooms.

Hydrocyanic acid gas is lighter than air and has an agreeable aromatic odor quite familiar in the flavoring essence of bitter almonds. The best method of generating it for the purpose of fumigation is by the action of dilute sulphuric acid upon potassium cyanid, in the following proportions:

Potassium cyanid	1.0 part
Sulphuric acid	1.5 parts
Water	2.25 parts

The first step is to dilute the acid, which is done by adding the acid to water in a vitrified clay jar or receptacle capable of withstanding the heat. The whole amount of cyanid must be put into the acid at once. As the evolution of the gas is very rapid, the operator should be ready to leave the spot immediately. As pointed out by Fulton, it is convenient to tie the cyanid up in a bag made of cheese cloth or tissue paper, which is lowered into the acid by a cord passing outside of the room. The amount of gas used for plant fumigation, expressed in terms of cyanid, is about 1 ounce per 100 cubic feet; about the same quantity is effective as an insecticide in rooms and confined spaces. Hydrocyanic acid gas is quite as effective as sulphur dioxid, is not destructive, is reasonably cheap, and is certain in its action, but its poisonous nature is such a serious drawback that it has a limited place as an insecticide in the disinfection of houses.

Bisulphid of Carbon.—Bisulphid of carbon (CS_2) is a very efficient insecticide, but a dangerous one, on account of its inflammable and explosive nature. It quickly kills mosquitoes, roaches, flies, ants, and insects of all kinds, as well as rats, mice, and squirrels. When pure it is a mobile, colorless liquid with an agreeable ethereal odor, but often it has a more or less fetid odor from the presence of other volatile compounds. The liquid must be kept in well-stoppered bottles in a cool place, and away from the light and fire. It evaporates rapidly at ordi-

nary temperatures, so that in using this substance in a confined space it is sufficient to pour it into open pans. Carbon bisulphid is very inflammable—more so than ether—and burns with a pale blue flame yielding sulphur dioxid and carbon dioxid or monoxid. In its use every precaution must be taken to see that there is no fire, lighted cigar, etc., in or about the field of operation. On account of its poisonous nature, if used in a house or other inhabited structure, the rooms must be thoroughly aired after its use.

According to Hinds, shallow tin pans or plates make good evaporating dishes for carbon bisulphid. The larger the evaporating area the better. About one square foot of evaporating surface is used to every 25 square feet of floor area, and one-half to one pound of the liquid carbon bisulphid is used for each square foot of evaporating surface. These figures, of course, are only suggestive and approximate. The pans should be placed as high in the room as possible, since the vapor is so heavy that it settles rapidly. Care should be taken when placing the pans to see that they are nearly level so as to hold the liquid, though ordinarily no particular harm will be done if some of it is spilled. It should not be found necessary to lose time in adjusting such things after the operation has begun.

Carbon bisulphid is being extensively used in California in the plague campaign. A piece of waste the size of an orange is saturated with the liquid and the wet ball placed in the mouth of the squirrel hole. Wet clay is then stamped into the warren so that the gas which is generating may have no opportunity to escape. All of the holes of the burrows are treated in this way. In some instances the ball is placed deeply in the hole and then ignited. This is more or less dangerous, as an explosion occurs, and, while the gas is thus disseminated to all parts of the warren, its action only covers a limited period of time, and is, therefore, not as certain as simply allowing the carbon bisulphid to evaporate. It not only kills the squirrels, but also the fleas on them. Carbon tetrachlorid may be used in place of carbon bisulphid. It is just as poisonous but neither inflammable nor explosive.

Petroleum.—Petroleum, kerosene, or coal oil is a very valuable insecticide, but of limited application, as it must be used in liquid form. As a remedy for mosquitoes it is applied in the proportion of about 1 ounce to 15 square feet of water surface. It should form a uniform film over the surface, and will then destroy the larvæ and pupæ of the mosquito and the adult females coming to the water to lay their eggs. The oil must be renewed every week or two, depending upon the temperature and other conditions. A light grade of fuel oil is best for this purpose (see page 203).

Petroleum is also useful against roaches, bedbugs, fleas, lice, and other insect vermin when used by direct application or by spraying,

either in the form of the pure oil or as an emulsion. Petroleum is very efficient against fleas. Frequent application to the floor or other places will keep away ants, and by direct application to the breeding, feeding, and traveling places it is a useful remedy against household vermin in general. By direct application to the head or other parts affected, coal oil is the cheapest and most effective remedy for lice.

Emulsion of crude petroleum for application to the skin of animals or to trees, or other plants, or for general insecticidal purposes is made from the formula of T. M. Price:

Crude petroleum	2 gallons
Water	½ gallon
Hard soap	½ pound

Dissolve the soap in the water with the aid of heat. To this add the crude petroleum; mix with a spray pump or shake vigorously and dilute with the desired amount of water. The emulsion of crude petroleum made according to this modified formula remains fluid, and can be easily poured. It will stand indefinitely without any tendency toward separation of the oil and water, and can be diluted in any proportion with cold soft water.

Arsenic.—The arsenical compounds, according to Marlatt,[1] have supplanted practically all other substances as a food poison for biting insects. The two arsenicals in most common use obtainable everywhere are arsenate of lead and Paris green. Scheele's green and arsenite of copper are less known and less easily obtainable, but in some respects are better than Paris green. The use of powdered white arsenic is not recommended on account of its corrosive action, as well as the fact that it is apt to be mistaken for harmless substances.

The arsenical poisons may be applied in one of three ways: (1) in suspension, as poisoned waters, mainly in the form of sprays; (2) as a dry powder blown or dusted about the infested areas; or (3) as poisoned bait.

It must be remembered that the arsenicals are very poisonous, and should be so labeled, and care taken to prevent accidents.

Paris green is a definite chemical compound of arsenic, copper, and acetic acid (acetoarsenite of copper), and should have a nearly uniform composition. It is rather a coarse powder, or, more properly speaking, crystal, and settles rapidly in water, which is its greatest fault so far as the making of suspensions of this substance is concerned. The cost of Paris green is about 20 cents per pound.

Scheele's green is similar to Paris green in color and differs from it only in lacking acetic acid; in other words, it is simply arsenite of

[1] *Farmers' Bulletin No. 19*, U. S. Dept. of Agriculture.

copper. It is a finer powder than Paris green, and, therefore, is more easily kept in suspension, and has the additional advantage of costing only half as much per pound.

Arsenite of lead is prepared by combining, approximately, 3 parts of the arsenite of soda with 7 parts of the acetate of lead (white sugar of lead) in water. These substances, when pulverized, unite readily and form a white precipitate, which is more easily kept suspended in water than any of the other arsenical poisons. Bought at wholesale, the acetate of lead costs about 7½ cents a pound, and the arsenite of soda costs about 7 cents a pound. Its use is advised where excessive strengths are not desirable, and upon delicate plants, where otherwise scalding is likely to result.

An average of one pound of either Paris green or Scheele's green, or London purple to 150 gallons of water is a good strength for general purposes in using the wet method. The powder should first be made up into a thin paste in a small quantity of water, and, if the suspension is to be used upon plants, vegetables, or about foliage, an equal amount of quicklime should be added to take up the free arsenic and remove or lessen the danger of scalding.

For the distribution of dry poison the arsenicals are diluted with 10 parts of flour, lime, or dry gypsum.

The following mixtures are used in the form of sprays, to destroy insects and fungi upon plants.[1] The arsenate of lead mixture has been much used in Massachusetts with success against the gipsy moth and other destructive insects upon trees and plants. These mixtures are equally useful as insecticides wherever sprays or local applications are practicable.

ARSENATE OF LEAD

Arsenate of soda (5 per cent. strength), 4 ounces.
Acetate of lead, 11 ounces.
Water, 100 gallons.

Put the arsenate of soda in 2 quarts of water in a wooden pail, and the acetate of lead in four quarts of water in another wooden pail. When both are dissolved, mix with the rest of the water. Warm water in the pails will hasten the process. For the elm-leaf beetle use 10 instead of 100 gallons of water.

A number of ready-made arsenates of lead are now on the market, and, except when very large amounts are needed, it will probably prove cheaper to buy the prepared material than to make it. With this ready-made material take 3 pounds to 50 gallons of water for codling moth, and 5 pounds to 50 gallons to the elm-leaf beetle and on potatoes.

[1] From *Bulletin No. 123*, April, 1908, of the Massachusetts Agricultural Experiment Station by Stone and Ferald.

ARSENITE OF LIME

White arsenic, 2 pounds.
Sal-soda, 8 pounds.
Water, 2 gallons.

Boil till the arsenic all dissolves—about 45 minutes. Make up the water lost by boiling and place in an earthen dish. For use take one pint of this stock, 2 pounds freshly slaked lime, and 45 gallons water, and spray.

KEROSENE EMULSION

Hard soap, shaved fine, ½ pound.
Water, 1 gallon.
Kerosene, 2 gallons.

Dissolve the soap in the water, which should be boiling; remove from the fire and pour it into the kerosene while hot. Churn this with a spray pump till it changes to a creamy, then to a soft, butter-like, mass. Keep this as a stock, using one part in nine of water for soft-bodied insects, such as plant lice, or stronger in certain cases.

RESIN-LIME MIXTURE

Pulverized resin, 5 pounds.
Concentrated lye, 1 pound.
Fish or other animal oil, 1 pint.
Water, 5 gallons.

Place the oil, resin and one gallon of hot water in an iron kettle and heat till the resin softens; then add the lye and stir thoroughly; now add 4 gallons of hot water and boil till a little will mix with cold water and give a clear, amber-colored liquid; add water to make up 5 gallons. Keep this as a stock solution. For use take:

Stock solution, 1 gallon.
Water, 16 gallons.
Milk of lime, 3 gallons.
Paris green, ¼ pound.

BORDEAUX MIXTURE

Copper sulphate (blue vitriol), 4 pounds.
Lime (unslaked), 4 pounds.
Water, 25 to 50 gallons.

Dissolve the copper in hot or cold water, using a wood or earthen vessel. Slake the lime in a tub, adding the water cautiously and only

in sufficient amount to insure thorough slaking. After thoroughly slaking, more water can be added and stirred in until it has the consistency of thick cream. When both are cold, dilute each to the required strength and pour both together in a separate receptacle and thoroughly mix. Before using, strain through a fine mesh sieve or a gunny cloth; the mixture is then ready for use.

If the amount of lime in the Bordeaux mixture is insufficient there is danger of burning tender foliage. In order to obviate this, the mixture can be tested with a knife blade or with ferrocyanid of potassium (1 oz. to 5 or 6 oz. of water). If the amount of the lime is insufficient, copper will be deposited on the knife blade, while a deep brownish-red color will be imparted to the mixture when ferrocyanid of potassium is added. Lime should be added until neither reaction occurs. A slight excess of lime, however, is desirable, and it is seldom one has to apply these tests.

The Bordeaux mixture is a good fungicide, but is less useful as an insecticide.

MOSQUITOES

Mosquitoes differ markedly in their habits. Some species may be classed as domestic animals because they are commonly or almost exclusively found in or close to human habitations. This is notably the case with *Stegomyia calopus*, the yellow fever mosquito; *Culex pungens*, the intermediary for *Filaria bancroftii* (filariasis); and *Culex fatigans*, the carrier of dengue fever. The sylvan or wild mosquitoes, of which the *Culex sollicitans*, the common salt marsh mosquito of our Atlantic coast, is a well-known example, are seldom met with in human habitations. A third or semi-domestic class may be encountered either in or near houses, or in fields or swamps. This class includes the malarial mosquitoes belonging to the genus *Anopheles*.

The adult mosquito may be carried to considerable distances by winds; but of its own volition it does not ordinarily travel outside of a radius of half a mile from its breeding place. Most species do not fly nearly so far. This means that the destruction of all breeding places within a comparatively small radius of a habitation will rid it of all but those mosquitoes which are blown in by the winds from more or less distant marshes, or which are brought in the vessels and vehicles of trade and travel.

Life History and Habits.—Mosquitoes pass through four stages: (1) the egg or embryo, (2) the larva, (3) the pupa, and (4) the imago or adult winged insect. The egg, larval, and pupal stages are aquatic. Mosquitoes never breed in damp grass, weeds, or bushes, as is popularly supposed, but the winged insects frequently rest and hide in vegeta-

tion. The different species of mosquitoes not only differ markedly in their habits, but differ considerably in the character of their breeding places. The domestic species, such as the yellow fever mosquito and *Culex pungens*, may be found breeding in any collection of water in or about houses. Thus, they have been found in discarded tin cans, bottles, and broken crockery on the garbage heap; in buckets, tubs, barrels, cisterns, and wells; in baptismal fonts; in flower pots and sagging roof gutters; in street and roadside puddles, gutters, and ditches; in cesspools and sewers.

The semi-domestic mosquitoes, to which the malarial-bearing insects belong, may occasionally be found breeding in tin cans, barrels, hoof prints, post holes, and hollows in trees or tree stumps, but they usually prefer grass-bordered pools, slowly flowing ditches, the margins of lakes and streams, even such as are stocked with fish, provided the margins are shallow or are more or less choked with reeds and water plants so that the fish cannot reach them. The sylvan or wild mosquitoes select breeding places of much the same character as do the semi-domestic species, with which they are not infrequently found associated, except that such breeding places are more or less remote from human habitations, in woods, swamps, and fresh or salt (brackish) coastal marshes.

Male mosquitoes are vegetarians. The females of many species have developed a taste for blood, and, indeed, blood has become indispensable to nearly all for the full development of their eggs. This is the case with *Stegomyia calopus*. Remembering how all-important the generative instinct is, we can now well understand why the yellow fever mosquito, for example, will, when disturbed, return again and again in an endeavor to obtain her fill of this life-giving fluid.

The mosquito lays her eggs upon the surface of the water, and these, depending upon the species, either float separately on their sides (*Stegomyia calopus* and *Anopheles*), or adhere together in irregular, raft-like masses (*Culex*). In a day or two, under ordinary conditions, the eggs hatch out into larvæ or "wiggle-tails." Although the larva is an aquatic animal, it is a true air-breather. The larva of Anopheles ordinarily rests and feeds at the surface, where it lies in an almost horizontal position, its tail and dorsal bristles touching the surface film, while it breathes through a breathing siphon, which is very short and insignificant in appearance.

The larvæ of the other species move about more or less, actively searching for food, but at intervals of a minute or two they may be seen to come to the surface for air, where they hang, head down, attached by their more or less prominent conical breathing tubes to the surface film. The mosquito remains in the larval stage about a week and is then transformed into a comma-shaped creature known as the pupa.

The pupa has no mouth and does not feed. It remains quietly at the surface except when disturbed. It breathes through a pair of trumpet-shaped tubes, which project from the dorsum of the thorax. The pupal stage usually lasts two or three days, and is terminated by the emergence of the adult winged insect (imago) from its pupal case through a rent in the region of the breathing tubes.

The time from the laying of the egg to the winged insect may, therefore, be as short as nine days. The time depends upon the temperature and the abundance of the food supply. Warmth favors and cold retards; therefore, mosquitoes are most abundant during the summer, late spring, and early fall months in our climate. In the tropics the wild species become more abundant during the wet season.

The way in which mosquitoes manage to pass through the rigors of the winter probably varies with the different species. Some, like the malarial Anopheles, hide in sheltered cellars or dark nooks, or hibernate in other out-of-the-way places. Other species survive through the power of the larva or egg to resist cold, for the larvæ or eggs of some species will hatch even after they have been frozen.

THE DESTRUCTION OF MOSQUITOES

The life of a mosquito may be divided into an aquatic and an aerial stage, the former including the egg, larva, and pupa, and the latter the adult winged insect. Accordingly, the measures aimed at the destruction of the mosquito naturally fall into two classes: (a) those directed against the larva and pupa—the aquatic stages—and (b) those directed against the winged insect.

For the extermination of mosquitoes the most effective measures are those which aim to destroy their breeding places, and thus prevent their multiplication. For the best results both individual and communal effort are necessary, but the importance of individual effort alone cannot be too much emphasized. The individual, by attacking the problem on his own premises, grounds, or estate, can not only do much to rid his own immediate neighborhood of mosquitoes, and thereby increase his own comfort and guard against disease, but the example thus set will perhaps stimulate his less enterprising neighbor.

To insure success it is important to know the habits and breeding places of the particular species that it is desired to suppress.

Natural Breeding Places.—Natural collections of water which may serve as breeding places are best dealt with by filling in or by draining. In this way they are disposed of once for all. For filling, inorganic refuse, such as cinders and ashes, may be employed, or sufficient earth may be dug from a nearby knoll or hill, care being observed that in so doing a depression capable of holding water is not made. Low marshy

lands adjacent to rivers, lakes, or the sea may be filled by pumping silt or sand.

When filling is not practicable, good and permanent results may be obtained by drainage. As a rule, the draining of ponds, pools, or marshes is the simpler and cheaper method. By the draining of marshes is meant the draining of the pools of stagnant water, or in the case of coastal marshes the draining of the stagnant fishless pools that are beyond the reach of the ordinary tides; it does not necessarily include the draining of the water-soaked soil itself. The underdraining of wide acreages of our arable land in the Middle West has been very effective in suppressing the malarial mosquito. Marshy lands may be drained simply by means of ditches. These must be dug of sufficient depth to completely empty the pools under treatment and have sufficient fall to prevent stagnation in the course of the ditch itself. Where a sufficient fall is not obtainable fishless pools may be connected with those containing fish or with a neighboring stream, so that the fish may freely enter. Mosquito breeding places in the pools in coastal marshes may be suppressed by connecting them with tide water, so that they may be freely scoured by the daily tides. Ditches should have straight sides and must be inspected at frequent intervals, and care must be taken to see that they do not become choked.

Fish are among the most effective of the natural enemies of the mosquito. The fish may be admitted to ponds and pools in the manner just described, or the ponds, pools, ornamental lakes, and fountains may be directly stocked with minnows or gold fish. The margins of pools, rivers, and other bodies of water must be kept free of reeds and water plants, so as to permit the fish to reach the edges—a favorite breeding place for mosquitoes. One of the very best means of clearing the land of the numerous small natural collections of water is to place it under cultivation.

When radical measures, such as filling in or draining, are not practicable, resort may then be had to coal oil. Coal oil upon the surface of the water acts mainly by suffocating the larvæ and pupæ. A light quality of oil should be used, and it may be poured upon the surface from an ordinary sprinkling pot, or the surface may be sprayed with a hose. Along the banks of ponds, lakes, and slowly moving streams with shallow margins containing vegetation, which offer favorite breeding places for the mosquito, the oil may be applied with a mop. This practice is laborious, but effective. Sufficient oil should be used to cover the entire surface with a thin film. As the oil is volatile, it may disappear within a few days. Furthermore, the film, which should be intact to be effective, may be broken by winds. A strong wind will blow all of the oil to one side, thereby entirely defeating the object desired. It is, therefore, important to repeat the oiling regularly at intervals

of not more than one week, and as often in addition as necessary. Oiling, though fairly effective when properly carried out, is only a temporary expedient, and in the end is rather expensive. (See also page 206.)

No body of water is too small for a mosquito nursery. They breed in puddles by the roadside; in water that accumulates in furrows in gardens or fields, especially in clayey soil; in street gutters and house gutters; in holes in rocks; in hollows in trees, and anywhere that half a pint of water is allowed to stand.

Artificial Breeding Places.—The permanent elimination of artificial breeding places for mosquitoes in a city depends first of all upon providing a good quality and sufficient quantity of portable water by means of a modern closed system. This will permanently do away with the necessity of cisterns, barrels, and tubs for the storage of water about the premises. When domestic storage is a necessity, care must be taken to prevent the mosquito from gaining access to the water. The water barrels should be provided with tightly fitting covers. Burlap, sheeting, or several thicknesses of cheese-cloth, or, better, wire screening held in place by a well-fitting hoop, serve this purpose very well. Wooden covers are unsatisfactory, for they rarely fit accurately enough to keep out the mosquito, and this defect is enhanced by the warping of the wood, which usually makes an old cover worse than useless. More satisfactory than the wooden cover is one made of light galvanized sheet iron, the central portion of which may be made of wire gauze. The rim of the barrel should be trimmed to remove any irregularities that might prevent the cover from fitting evenly all around. Whatever the form of the cover employed, it should not be removed except for cleaning or refilling the barrel. The water should be drawn from a spigot. Where the water is very turbid and must undergo sedimentation before being used, several barrels should be provided for its storage and the water used from each barrel in turn. In such a case the spigot should be placed about a foot from the bottom, so that the sediment need not be disturbed as the water is drawn off for use. Wells should be provided with tight covers and the water drawn by pumps.

Cisterns and tanks should also be provided with accurately fitting covers, and should be inspected frequently for seams and cracks resulting from warping and shrinking of the wood. To guard against this loophole, wire gauze should be used to screen the joint between the tank and its cover. The gauze should include about one foot of the tank and overlap well upon the cover. The inlet to the tank or cistern should be provided with a cap of copper meshed wire gauze which may be protected by another and coarser meshed cap of stout wire, to prevent its choking with leaves, etc. As an additional precaution, the inlet pipe should be long and extend well below the water level. In cases of emergency, as in times of epidemics of yellow fever or dengue, where

the permanent measures for preventing mosquito breeding have been neglected, the surface of the water in barrels, tanks, and cisterns may be covered with some neutral non-volatile oil which does not impart a taste to the water.

Cesspools and privy vaults should be done away with and replaced with dry earth closets or a water carriage cistern. Where this has not been done they may be frequently and copiously oiled.

Among the artificial breeding places for mosquitoes may be mentioned chicken-pens in poultry yards; water cups on the frames of grindstones; baptismal fonts; tin cans or broken bottles in back yards; the catch basins of sewers; the water that stands in sagging house gutters; flower-pots, and similar places.

Screening.—Mosquito screens are the obvious and most effective single measure for personal prophylaxis where disease-carrying mosquitoes exist. In order to be effective the screening must be intelligently carried out with careful attention to details. The screen itself must be sufficiently close to keep out the mosquitoes. Some of them are able to squeeze through surprisingly narrow chinks. I was able to demonstrate, in the experimental work at Vera Cruz, that the stegomyia mosquito can pass a metal wire screen containing 16 strands or 15 meshes to the inch, but cannot pass one containing 20 strands or 19 meshes to the inch. When the screen consists of a fabric which is apt to pull out of shape so that some of the meshes are larger than others, it is advisable to use a net woven closer than 20 strands to the inch. Experience in malarial and yellow fever districts has taught this lesson, so that it is customary in those countries to use a rather closely woven material resembling nainsook. Metal screens made of iron wire are cheapest only when first cost is considered. They hardly last a season unless painted, in which case the size of the mesh is considerably reduced and interferes with ventilation, a serious objection in hot weather or a tropical climate. Mesh made of galvanized iron wire has a greater durability. Screens made of brass or bronze are expensive, but cheap in the long run, as they are found to last almost indefinitely.

The screening should include the entire house, or at least those parts that are occupied. In the tropics it is better to screen the galleries than each individual window. In any case, frequent and repeated inspection should be made to discover breaks in the screen or openings due to warping of the woodwork. In screening care must be exercised not to overlook fireplaces, ventilators, and other openings. The door should be guarded by a screened vestibule of such a depth as to make it impossible for a person to hold both doors open at the same time. The screen door should open outward and, if possible, should be exposed to the direct sunlight during the day without vines or nearby vegetation of any kind to protect and lodge the mosquitoes. During the night

the door should not be in an artificial light, which attracts many mosquitoes. An electric fan directed outward is a very good device to prevent mosquitoes flying through the doorway. In addition, a whisk-broom or feather duster should hang in the vestibule to brush off the insects that may rest upon the clothing. A screened house is safe only to careful and intelligent people.

In addition to screening the house, mosquito bars over the bed will be found necessary in mosquito-infected places. It is best to suspend the mosquito bar from the ceiling and carefully gather the bottom together so as to keep the insects out during the day time. At night the bar should be carefully tucked in around the bed so as to leave no openings. Mosquitoes have no trouble in biting through the meshes of the bar, provided a restless sleeper comes close enough to it.

Persons who are required to go out at night in a malarious district, or who must expose themselves during yellow fever times, may screen themselves effectively with a veil of mosquito netting hanging from a broad-brimmed hat to the shoulders and chest. The hands and wrists may be protected with gloves, and the ankles with leggings or other suitable mechanical device.

Miscellaneous Measures.—Spirits of camphor, oil of pennyroyal, and other volatile substances, such as oil of peppermint, lemon juice, or vinegar, rubbed upon the face and hands, or a few drops on the pillow at night, will keep mosquitoes away only for a time. Oil of citronella is one of the best known substances to be used in this way. Ordinarily a few drops on a bath towel hung over the head of the bed will keep the common house mosquitoes away. When they are very abundant and persist, a few drops rubbed on the face and hands will suffice. All these substances soon lose their efficiency; none of them last until morning.

In Panama a larvicide is being used which is made as follows: 150 gallons of carbolic acid is heated in a tank to a temperature of 212° F., then 150 pounds of powdered or finely broken resin is poured in. The mixture is kept at a temperature of 212° F. Thirty pounds of caustic soda is then added, and the solution is kept at the same temperature until a perfectly dark emulsion without sediment is formed. The mixture is thoroughly stirred from the time the resin is used until the end. One part of this emulsion to 10,000 parts of water is said to kill Anopheles larvæ in less than half an hour, while 1 part to 5,000 parts of water will kill them in from 5 to 10 minutes.

The Panama larvicide is mixed with 5 parts of water and sprayed upon pools or along the banks of streams. This larvicide added to 5 parts of crude petroleum favors its spread upon the surface of the water. A good method is to place the mixture in a barrel and permit it to drip upon the surface of the stream or pond to be treated.

Other larvicides that may be used in water not used for drinking

purposes are: sulphuric, hydrochloric, and other acids, potassium permanganate, sulphate of copper, sulphate of iron, bichlorid of mercury, carbolic acid, anilin products, or coal tar. They must be used in relatively large amounts to be effective, and frequently renewed according to circumstances.

The diseases known to be conveyed by mosquitoes are: malaria (*Anopheles spp.*), yellow fever (*Stegomyia calopus*), filariasis (*Culex fatigans*), dengue (*Culex fatigans*), and doubtless other infections.

MALARIA

Malaria is one of the most prevalent of all preventable diseases; it is the scourge of the tropics. The cause of this infection was one of the first to be discovered (Laveran, 1880), and its mode of transmission was one of the most brilliant discoveries in sanitary science (Ross, 1895). Despite the fact that we have more exact knowledge of malaria, considering the difficulties of the subject, than perhaps any other disease, despite the fact that we have accurate means of diagnosis and a ready cure, and despite the fact that we have assured measures of prevention, malaria counts its victims by the hundreds of thousands annually. In geographic distribution malaria extends from the Arctic circle to the Equator, but becomes more virulent the warmer the climate.

At least three separate malarial parasites of man are known, namely: (1) *Plasmodium malariæ* (Laveran), quartan fever; (2) *Plasmodium vivax* (Grassi and Filetti), tertian fever; and (3) *Plasmodium falciparum* (Welch), estivoautumnal or tropical malaria. These are closely allied hematocytozoa or blood parasites. They produce diseases with well-defined clinical differences, but having the same etiology and mode of transference, so that, as far as prevention is concerned, they may be regarded as one infection.

Many species of animals have a malarial-like infection closely resembling malaria in man; for example, Texas fever of cattle, piroplasmosis of dogs and sheep, proteosoma of birds, etc. So far as is known, no other animal than the Anopheles mosquito is subject to the malarial parasites pathogenic for man. Both man and the mosquito are necessary to complete the life cycle of the plasmodium. Man is the intermediate host harboring the asexual phase, and the mosquito is the definitive host harboring the sexual phase of the life cycle of the plasmodium.

Mosquito Transmission.—It is now definitely known that in nature malaria is transmitted only by the sting of the Anopheles mosquito.[1] Experimentally, the infection may be transferred by injecting blood

[1] The genus Anopheles has recently been divided into several genera.

(containing the parasites) of one person into the system of another. Nearly 2,000 years ago Varro and Columbella mentioned the possibility that the disease was transmitted by mosquitoes. In Africa some savage tribes call malaria the "mosquito disease." In 1848 Nott, of New Orleans, considered the matter proven from biological analogies. In 1882 King, of Washington, vigorously advocated the mosquito theory based upon philosophical deductions but no proof. In 1884 Laveran suggested mosquito transmission as probable. In 1894 Manson elaborated the mosquito theory and inspired Ross, of the Indian Army Medical Service, who in 1895 demonstrated that the crescents of estivoautumnal malaria underwent changes in the mosquito. In 1896 Bignami advocated the theory and compared it to the transmission of Texas fever by the tick. In 1897 Ross published further convincing observations upon the development of the estivoautumnal parasite in the mosquito. In 1898 McCollum observed an important missing link in the life cycle by observing the flagellum of the microgametocyte (male) fertilize the macrogametocyte (female) with the formation of the vermicule. These observations were made upon Halteridum or malaria of birds; later he saw the same phenomenon in estivoautumnal malaria. The life cycle of the malarial parasite has been confirmed by Daniels, Koch, Grassi, Bignami, Celli, Manneberg, Schaudinn, and others.

Further evidence that malaria is transmitted by the mosquito was furnished by Sambon and Low, of the London School of Tropical Medicine, and Dr. Terzi, who lived during the three most malarial months of 1900 in Ostia, a very malarial locality of the Roman Campagna. These observers escaped infection simply by keeping within their well-screened hut from before sundown until after sunrise. The final proof was furnished in 1900 by Dr. P. Thurber Manson and Mr. George Warren, who were bitten by infected mosquitoes forwarded from Italy in cages to London.

The Malarial Mosquito.—Of the fifty or more species of the genus *Anopheles* sixteen are known to transmit malaria. In Europe *Anopheles maculipennis,* in tropical America *A. argyrotarsus* or *albipes,* in temperate America, *A. quadrimaculatus,* which is probably the same as *A. maculipennis,* in India *A. sinensis,* in Africa *A. costalis,* are the chief culprits.

The Anopheles mosquitoes are brownish and rather large. They may be distinguished by the fact that the palpi in both the male and the female are at least as long as the proboscis. Only the female transmits the infection. It sits more or less at right angles upon the wall, the head, thorax, and abdomen being in a straight line. Contrary to the yellow fever mosquito, the malarial mosquito is nocturnal in its habits and breeds chiefly in the open ponds, puddles, and natural collections of water in the woods, fields, and swamps.

The mosquito becomes infected upon drinking the blood containing the micro- and macrogametocytes. It requires about twelve days before the sporozoites appear in the salivary glands of the insect. It cannot, therefore, transmit the infection to another person until the lapse of this extrinsic period of incubation. The infected mosquito may live a long time and infect more than one person successively. The malarial parasite seems to be a harmless saprophyte for the mosquito.

Fig. 19.—Anopheles Punctipenis.

Immunity.—A person who once has had malaria is more apt to have subsequent attacks. Ordinarily there is an increased susceptibility rather than an immunity. However, repeated infections, especially during early life, leave a very pronounced resistance. In malarious regions many children carry the parasites in their circulating blood without any manifestations of the disease. These carriers are important factors in spreading the infection in endemic areas, and must be taken into account in preventive measures.

There is no true racial immunity in this disease. Occasionally a congenital immunity seems to be transmitted; this must be rare. Practically all persons who receive the infection for the first time are susceptible. The freedom from malaria which some persons seem to enjoy may be accounted for partly by the fact that mosquitoes seldom bite such persons. It is well known that on account of the odors, or what not, mosquitoes do not bother certain individuals. No doubt the infection of a small number of parasites is often overcome largely through a vigorous phagocytosis.

Individual resistance varies in different individuals and in the same individual at different times. The parasite may remain latent in the spleen and other organs for years. Exposure, overeating, fasting, overwork, or worry, or anything that lowers the vitality of such individuals predisposes to an attack of malaria. The disease often breaks out in persons in good health leaving a malarial region for a health resort, whether mountain or seashore. I was enabled to confirm this observation upon the returning transports from Cuba following the Spanish-American war, when many cases of malaria broke out among the troops previously in good health upon reaching the cold winds about Cape

Hatteras. Personal prophylaxis, therefore, involves careful attention to personal hygiene.

Prevention.—The successful suppression of malaria requires a combined attack upon the mosquito and the parasite in the human host. Ultimate success rests upon the suppression of the mosquito. This, however, is a difficult and expensive undertaking in the case of the Anopheles. Immediate relief is most quickly gained by measures directed against the infection in man. Screening and quinin prophylaxis, while practical, are only temporary measures.

Measures Directed Against the Mosquito.—If the breeding of the Anopheles mosquito could be stopped malaria would cease. Mosquito suppression is fundamental and radical. The best way to abolish the breeding places of malaria mosquitoes is to fill up low places or to dry the surface of the land with drains. These two measures hold first place as permanent work. The underdraining of large areas of our arable land of the Middle West with tiled drain has been very effective in suppressing malaria. Open ditches properly constructed and cared for are likewise effective. In the tropics the ditches should be lined with cement, on account of the luxuriant vegetation which soon interferes with their efficiency or may actually convert them into breeding places. The open ditches are much the cheapest in first cost, but not when maintenance is reckoned. The draining of swampy lands is an engineering problem in which the economic factor looms large. One of the very best means of destroying the breeding places of the malaria mosquito is to clear the land and to keep it in cultivation.

When drainage is not practical, the number of mosquitoes may be kept down by introducing fish into the pools, streams, ditches, and other collections of water. Upon limited water surfaces the larvæ may be killed with a film of coal oil.

Large open spaces cause the destruction of a number of mosquitoes, as they cannot live long in the hot sun; therefore, clearing the brush and high grass, which furnish shelter to the insects, aids in keeping away wild mosquitoes around dwelling houses.

The use of screens and culicides has already been referred to.

Personal Prophylaxis.—Persons visiting or residing in a malarious region should be particularly careful not to expose themselves at night time. The experience of Sambon and Low on the Roman Campagna is instructive and should be imitated. The location of the residence is important. In a city it should be a reasonably safe distance from the native quarter, because the infection is there most concentrated. The dwelling should, if possible, face the trade winds. A row of tall trees will partly screen the house from the swamp, but the trees must not be too close, else they will furnish shelter for the insects. The house should be on high land if practicable, as it is an old observation

that the malarial mosquito does not fly high. People living upon the second floor are less apt to contract the infection than those who sleep on the ground floor. If it is necessary to go out in the night time, one may protect himself by the use of gloves and mosquito netting hanging from the helmet to the shoulders. Care must be taken to guard the ankles against mosquito bites. As all these measures require much time and attention to details, they are usually not sufficient in actual practice. Therefore, quinin prophylaxis is much used.

Quinin Prophylaxis.—Theoretically the administration of quinin to healthy individuals for the prevention of malaria is not an ideal method of prophylaxis, for it does not prevent infection, but only destroys the parasites in the blood during the period of incubation. It should be remembered that quinin kills only the young and tender forms of the plasmodium, and has no influence upon the crescents. Quinin prophylaxis is indicated in proportion to the difficulty of pursuing more permanent methods. It is especially valuable where screens and bars are not available, as in camping, marching, traveling, or where the occupation takes one out at night. When residents of non-malarial countries go into malarial localities, especially in the rural districts, for short periods of time, quinin is a valuable preventive.

To be effective as a preventive of malaria, quinin must be taken in sufficient doses during the entire malarial season. The expense of public prophylaxis with quinin on a large scale is enormous; in fact, in some instances prohibitive. The daily ingestion of 2.5 grains would require the annual use of no less than 59.4 tons per million people. The size of the dose and the interval at which the prophylactic is administered are of the utmost importance. Koch advised one gram of quinin every sixth or seventh day, or every seventh and eighth day, or eighth and ninth, or ninth and tenth day, according to the danger of the infection. This manifestly leaves several intervening days in which there is no quinin in the circulation. In localities, therefore, where estivoautumnal malaria is prevalent, a shorter interval should be preferred on account of the shorter period of incubation of this form of malaria.

Ziemann gives a gram of quinin sulphate every four days. The alkaloid is administered in solution with 5 drops of hydrochloric acid early in the morning or about one and one-half to two hours after a meal. A convenient rule is to give a dose on the first of the month and thereafter on each day of the month divisible by 4. By this method the alkaloid is probably constantly in the circulating blood.

Plehn advises one-half a gram of quinin every fifth evening.

The administration of small doses of quinin daily is the oldest method of giving quinin as a prophylactic. From 1½ to 6 grains have been given daily. In Italy 0.04 gram (about 2-3 grain) daily is the universally

adopted dose, and accomplishes good results. The Italian government undertakes the sale of quinin at a low price. This is a beneficent public health measure comparable to the free distribution of antitoxin and vaccine virus.

On the Isthmus of Panama good results have been obtained by the use of moderate doses, 3 to 6 grains per day. When the disease increases in prevalence or virulence the amount is raised to 8 or 10 grains per day, then dropping off to 4 or 5.

The particular method of election in giving quinin prophylaxis should be chosen according to the experience of the region.

An objection to the use of quinin as a prophylactic has recently been raised by Stitt, who claims that the malarial parasites gradually become immune to the effects of the alkaloid, and that when the disease subsequently breaks out in one who has used quinin as a prophylactic it is not readily amenable to treatment. Ehrlich has shown experimentally that trypanosomes may be immunized in this sense to trypanrot, and that other microparasites belonging to the animal kingdom may similarly be accustomed to unusual amounts of substances ordinarily very toxic.

Quinin prophylaxis has advantages that commend it as a prompt and practical measure. It is at best, however, only tentative, and does not take the place of mosquito suppression.

YELLOW FEVER

The prevention of yellow fever rests entirely upon the fact that it is communicated through the bite of an infected mosquito—the *Stegomyia calopus*.[1] The mosquito becomes infected by sucking the blood of yellow fever patients during the first three days of the fever. All the experimental evidence thus far shows that the infection is absent from the blood after the third day, and that mosquitoes do not become infective after this period. The importance of this fact in preventing the spread of the disease is evident. The mosquito, after drinking the infected blood, is not able to transfer the infection to another person until about twelve days[2] have elapsed; that is, it requires about twelve days for the yellow fever parasite, whatever it may be, to undergo its cycle of development in the mosquito. The mosquito once infected remains so during the rest of its life, which may be many months. Only the female mosquito transmits the infection; the male *Stegomyia calopus* is a vegetarian; its proboscis is too soft to penetrate the skin. A single

[1] This mosquito was first called *Culex fasciatus*, which was changed to *Stegomyia fasciatus*, and then to *Stegomyia calopus*, and recently expressed as *Aëdes calopus* by Coquillett.

[2] This constitutes the extrinsic period of incubation, in contradistinction to the intrinsic period of incubation, that is, the time between the mosquito bite and the onset of symptoms, which is from 2 to 5 and sometimes 6 days in this disease.

sting of a single infected mosquito is sufficient to produce the disease. An infected mosquito may infect more than one person at different times.

The prevention and control of yellow fever are based upon a series of epoch-making investigations and discoveries (1900-1902) by a commission composed of Walter Reed, James Carrol, Aristides Agramonte, and Jesse W. Lazear, medical officers of the United States army. These experiments have been fully confirmed, and in some respects amplified, by independent workers, namely, Guiteras of Cuba (1901); Barreto, de Barros, and Rodrigues, of Brazil (1903); Ross (1902); Parker, Beyer, and Pothier (1903); Rosenau, Parker, Francis, and Beyer (1904); Rosenau and Goldberger (1906), of America; Marchoux, Salimbeni, and Simond (1903); Marchoux and Simond (1906), of France; and Otto and Neumann (1905), of Germany.

The cause of yellow fever is unknown. The virus is ultramicroscopic, that is, passes the close-grained pores of the finest porcelain filter. While in nature the disease is transmitted only through the bite of an infected Stegomyia, the disease may be transferred experimentally by taking some of the blood from a patient during the first three days of the fever and injecting it into a susceptible individual. So far as is known, yellow fever is peculiar to man, for all other animals tested have failed to react. At one time it was generally believed that yellow fever infection was conveyed by fomites. This has been disproved, and we now know that there is no danger from soiled clothing or other inanimate things, even though stained with the black vomit and other discharges.

The diagnosis of yellow fever rests upon clinical evidence and is frequently difficult to make, especially in the early stages. It is, therefore, important to screen all cases of fever in a yellow fever campaign until the nature of the illness is established.

Immunity.—There is no natural immunity to yellow fever. All persons receiving the infection for the first time seem to be susceptible. Contrary to the usual statement, there is no racial immunity in this disease, for negroes, Chinese, Indians, and other races take the disease. One attack of yellow fever affords protection against a subsequent attack. The acquired immunity in this disease is one of the strongest known and lasts throughout the lifetime of the individual. Two attacks of yellow fever are almost unknown. I reported a supposed instance in a Spaniard in Havana, but the diagnosis of the first attack was not conclusive.

In endemic areas children may have yellow fever, which leaves them immune for life. The disease often runs a mild and unrecognized course in children, and this fact explains the supposed natural immunity of natives in endemic foci.

The Yellow Fever Mosquito.—The yellow fever mosquito has a wide

distribution ranging from 38 degrees south to 38 degrees north latitude. They are found in the East and West Indies, China, Sumatra, Java, India, Philippine Islands, Japan, Hawaiian Islands, in the southern part of Italy, Africa, Spain, South America, etc. They usually prefer the lowlands. I have found them as far up the mountains as Orizaba in Mexico, 4,200 feet above sea level. In the United States they are very prevalent south of the Potomac along the gulf coast, but are absent or rare in the higher elevations of Georgia or Alabama, which are, therefore, non-infectable regions.

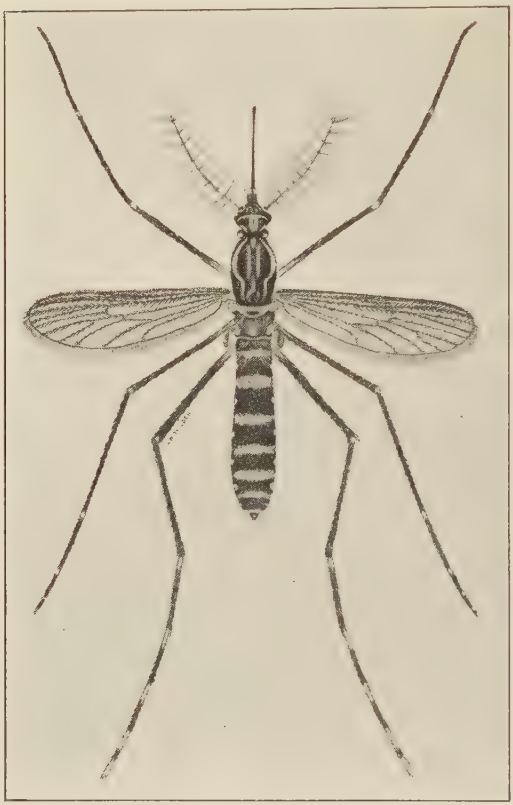

Fig. 20.—Stegomyia Calopus (female).

The yellow fever mosquito is a domestic insect. It breeds by preference in any standing water about the household, such as cisterns, rain barrels, or any collection of water in buckets, bottles, old cans, etc. The yellow fever mosquito does not breed in the fields, woods, and swamps, which are the favorite resorts of the malarial mosquito. The Stegomyia mosquitoes do not fly far of their own volition, but show a cat-like tendency to remain about their place of birth or adoption. All these facts have an evident bearing upon preventive measures. A thorough knowledge of the biology of the mosquito is essential to the success of a yellow fever campaign.

It is important to remember that the yellow fever mosquito is chiefly active during the day time. It cannot, however, distinguish between artificial light and sunlight. I have watched Stegomyia mosquitoes bite me by electric light at eleven o'clock at night. However, as a rule, they rest at night, which, therefore, diminishes the risk of exposure at that time. The Stegomyia mosquito, however, cannot survive for long in the direct rays of a tropical sun. There is, therefore, little

danger in visiting a community where yellow fever is epidemic during the day time, provided the person keeps out of houses. The experiences during the last yellow fever epidemic at New Orleans, 1905, showed that the radius of activity of an infected Stegomyia is contracted. It may possibly at times fly across the street, but it is evident that it neither flies far nor is it ordinarily transported to any great distance on railroad cars, although it may be carried over seas on ships.

The yellow fever mosquito may pass a screen composed of 16 strands or 15 meshes to the inch, but cannot pass one containing 20 meshes or 19 strands to the inch. Effective screens must, therefore, be at least this fine.

Fig. 21.—Head of Stegomyia Calopus (male).

Stegomyia calopus is a grayish mosquito of average size with beautiful glistening silver-white markings. These markings are lyre-shaped on the back of the thorax; silver-white spots are seen on the side of the thorax. White lines are apparent at each tarsal joint and also on the palpi; the scutellum is white. In the female the palpi are much shorter than the proboscis, which at once distinguishes it from Anopheles.

Egg.—The female lays her eggs on the surface of the water or just above the water line. The eggs do not adhere to one another, and hence do not form the compact boat-shaped mass characteristic of the culex, but float on their sides more or less singly. At the moment of laying the eggs are a cream color, but rapidly become jet black. They are somewhat cigar-shaped, and measure on the average about 0.55 mm. in length and 0.16 mm. in width at the broadest part. The eggs show marked powers of resistance to unfavorable influences. They may be kept dry for six and one-half months, and still retain their vitality, and hatch out when put back into the water. Freezing does not kill them. The egg probably plays an important rôle in the hibernation of the yellow fever mosquito. The winged insect may also survive a short winter. Under the most favorable conditions as to temperature (30° C.) Stegomyia eggs hatch out in about 36 hours after they are laid. Under 20° C. they will not hatch at all.

Larva.—The egg hatches the larva ("wiggle-tail"), which has a black barrel-shaped respiratory siphon. This distinguishes it from *Culex*

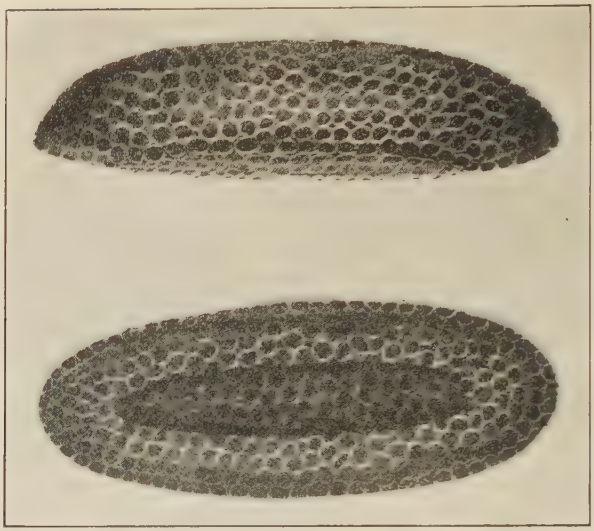

FIG. 22.—EGGS OF STEGOMYIA CALOPUS.

pipiens, its common mess mate, in which the air tube is brown, longer, and more slender. Although the larva lives in the water, it is strictly an air-breather and must come to the surface for air. It thrusts its breathing tube up into the surface film and remains suspended, head down, at an angle of somewhat less than 45 degrees, which distinguishes it from Anopheles larvæ, which lie horizontal. A film of oil on the surface of the water is sufficient to obstruct the air tube and thus cause

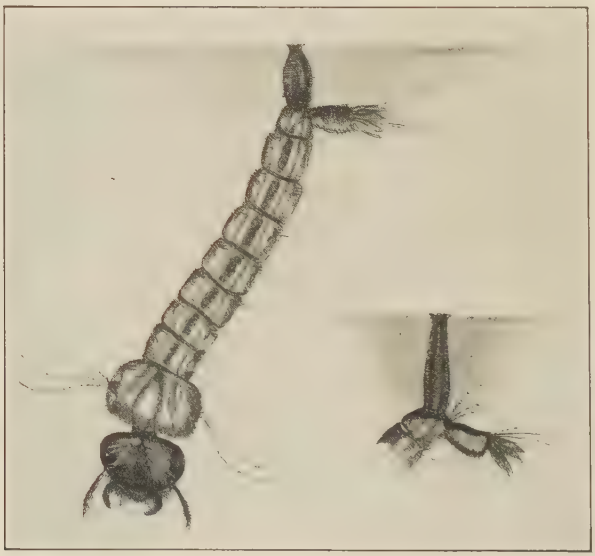

FIG. 23.—LARVA OF STEGOMYIA CALOPUS.
RESPIRATORY SYPHON OF CULEX TO THE RIGHT.

the death of the larva by suffocation. The larva is very timid, so that a slight jar or agitation or a sudden shadow will cause it to wriggle rapidly to the bottom, where, indeed, it may very commonly be observed to feed. The duration of the larval stage is never less than 6 to 7 days, and depends upon the food supply and temperature. Under favorable conditions it may be prolonged for weeks. Freezing for short periods does not appear to injure it.

FIG. 24.—PUPA OF STEGOMYIA CALOPUS.

PUPA.—The larva changes into the pupa. The pupa is not provided with a mouth and does not feed. It is an air-breather and spends most of its time at the surface of the water. The pupal stage lasts at least 36 hours, during which time metamorphosis occurs into the imago or perfect winged insect.

IMAGO.—Under the most favorable conditions it is at least 9 days from the time the Stegomyia lays its egg to the appearance of the imago. Under natural conditions the length of life of the adult female probably varies greatly. Guiteras succeeded in keeping a presumably infected one alive for 154 days during the fall and winter temperature in Havana. Deprived of water, it does not usually survive longer than 3½ to 4 days, and only very exceptionally 5 days. This fact has a bearing on the possibility of transporting the mosquito in band-boxes, trunks, and other containers.

"Aerial" Conveyance.—It is notorious that yellow fever is usually conveyed but a short distance "aerially"—perhaps across the street, or,

more often, to a neighboring house in the rear. This represents a distance of some 75 yards, which is about as far as we may expect it to be thus conveyed, from our knowledge of the habits and flight of the Stegomyia mosquito. The longest distance recorded in recent years of aerial conveyance is one of 225 meters (Melier) and one of 456 feet (Carter). These are entirely exceptional. My experience in the detention of hundreds of susceptible immigrants in quarantine for days in Havana harbor showed that infected Stegomyiæ do not travel a short distance across the water. This observation is in confirmation of others, that vessels moored within 1,200 feet of the shore are entirely safe so far as yellow fever is concerned, provided, of course, personal intercourse is interdicted or supervised.

Prevention.—The prevention or suppression of yellow fever may be attacked in either one of its two hosts, man or insect. If every person developing yellow fever were immediately isolated from the Stegomyia mosquito, the disease would inevitably cease. The elimination of the Stegomyia mosquito would give the same happy result. Usually both methods of attack are employed. It would seem easier to control the human host simply by screening during the first three or four days of the fever. Practically this method has been found insufficient, because the disease is difficult to diagnose in the early stage, and the mild cases escape attention. The essence of yellow fever prevention, therefore, consists in: (1) screening cases of yellow fever and all suspected cases of yellow fever; (2) destruction of infected insects; (3) the suppression of stegomyiæ through the control of their breeding places. It was a combination of these three methods which was first so brilliantly carried out by Gorgas in Havana in 1901, and later in Panama; by White in New Orleans, 1905; by Liceaga for Vera Cruz, and recently by Oswaldo Cruz in Rio de Janeiro, 1909.

Yellow fever patients should be isolated only in the sense of separating them from *Stegomyia calopus*. This may be done by proper screening. It is not necessary to remove the patient to a hospital, although this is desirable, for the reason that a special hospital is more carefully guarded than is practicable in a private house, and the trained assistants are an additional safeguard. As soon as the patient is removed, the mosquitoes in the house and the surrounding houses should at once be destroyed. Yellow fever patients must be moved with caution, for the reason that undue excitement or exertion seems to increase the severity of the disease.

The insecticides best suited for the destruction of mosquitoes are: sulphur dioxid, hydrocyanic acid gas, pyrethrum powder, tobacco smoke, Mim's culicide (camphor and phenol) (see page 187). At first glance it might appear to be a hopeless task to attempt to eradicate the yellow fever mosquito in a large city, but that this is possible was demon-

strated in New Orleans in 1905, when, after several months of a vigorous campaign, it was difficult to find a Stegomyia mosquito. The measures consisted mainly in screening the water cisterns and eliminating all standing collections of water in and about the household.

Historical Note.—Dr. Charles J. Finlay studied the relation of the mosquito to yellow fever as far back as 1882 and 1883. The first insects used by the United States Army Commission to bring about the demonstration of the new doctrine were received from the hands of Dr. Finlay. Finlay believed that the cause of the disease was a micrococcus and considered that the insects were capable of transmitting the disease a few days after they had stung a yellow fever patient. Sternberg's studies upon yellow fever are published by the Government as a report of the United States Marine Hospital Service on the Etiology and Prevention of Yellow Fever, 1890. Carter's observations at Orville, Mississippi, upon the extrinsic period of incubation were published in the *Medical Record,* June 15, 1901.

The work of the United States Army Commission appeared in the following publications:

"The Etiology of Yellow Fever—a Preliminary Note," *Proceedings* of the 28th Annual Meeting of the Am. Pub. Health Assn., Oct. 22-26, 1900; also *Philadelphia Med. Jour.,* Oct. 27, 1900.

"The Etiology of Yellow Fever—An Additional Note," *J. A. M. A.,* Feb. 16, 1901.

"Experimental Yellow Fever," *Am. Med. Jour.,* July 6, 1901.

"Etiology of Yellow Fever—Supplemental Note," *Am. Med. Jour.,* Feb. 22, 1902.

On account of their historical interest and accuracy, the conclusions of the United States Army Commission are here given:

1. The mosquito—*C. fasciatus*—serves as the intermediate host for the parasite of yellow fever.

2. Yellow fever is transmitted to the non-immune individual by means of the bite of the mosquito that has previously fed on the blood of those sick with this disease.

3. An interval of about 12 days or more after contamination appears to be necessary before the mosquito is capable of conveying the infection.

4. The bite of the mosquito at an earlier period after contamination does not appear to confer any immunity against a subsequent attack.

5. Yellow fever can also be experimentally produced by the subcutaneous injection of blood taken from the general circulation during the first and second days of the disease.

6. An attack of yellow fever produced by the bite of the mosquito

confers immunity against the subsequent injection of the blood of an individual suffering from the non-experimental form of this disease.

7. The period of incubation in thirteen cases of experimental yellow fever has varied from forty-one hours to five days and seventeen hours.

8. Yellow fever is not conveyed by fomites, and hence disinfection of articles of clothing, bedding, or merchandise, supposedly contaminated by contact with those sick with this disease, is unnecessary.

9. A house may be said to be infected with yellow fever only when there are present within its walls contaminated mosquitoes capable of conveying the parasite of this disease.

10. The spread of the yellow fever can be most effectually controlled by measures directed to the destruction of mosquitoes and the protection of the sick against the bites of these insects.

11. While the mode of propagation of yellow fever has now been definitely determined, the specific cause of this disease remains to be discovered.

Prevention of Malaria and Yellow Fever Contrasted.—For the prevention of malaria the same principles guide us that have been set forth for the prevention of yellow fever. In practical application, however, our methods of attack differ, owing to differences in the habits of the two mosquitoes, and owing to differences in the two diseases. The malarial problem is much more difficult, because it is harder to get rid of Anopheles than of Stegomyia. The breeding places of the yellow fever mosquito are practically confined to artificial containers in the neighborhood of human habitations, while those of anopheles are found in marshes, pools, or streams, and often in collections of water in the grass or brush. The breeding places of the malarial mosquito cover a much larger area, frequently the whole country, and are rather hard to find and difficult to destroy; also this insect travels much further from its breeding place than the Stegomyia, probably from three to four times as far. Compared to yellow fever, the control of the malarial human host presents special difficulties. In yellow fever man is infective to the Stegomyia only a few days; in malaria the parasites continue in the circulating blood a very long time. In the case of malaria, then, we have to deal with chronic carriers, which, fortunately for us, does not occur in yellow fever. For malaria we have quinin as a prophylactic, whereas no known drug will prevent yellow fever.

DENGUE

All who visit the tropics or subtropical countries where dengue prevails are very apt sooner or later to contract this infection. So far as known, few persons have ever died of dengue. Although the mortality

is practically nil, the disease is a painful affection and sometimes leaves the body in a weakened condition for long periods of time. In its epidemiology and symptomatology the disease strikingly parallels yellow fever, which adds to its importance. Outbreaks of dengue often precede and may be coincident with those of yellow fever. In the tropics influenza and dengue are also frequently confused. Dengue also has some resemblance to the three-day fever or pappataci fever of Herzegovina, which is transmitted by the bite of the *Phlebotomus pappatasii*, a biting fly.

There is no definite immunity produced by an attack of dengue. Persons often give a history of an attack in each outbreak. The cause of the disease is not known.

Graham studied dengue in Beirut, Syria, and described a protozoon inhabiting the red blood corpuscles and closely resembling the plasmodium of malaria except for the absence of pigment.[1] Graham believed that this organism underwent a developmental stage within the mosquito (*Culex fatigans*). He claimed to have observed the spores of this organism "in among the cells of the salivary glands" after 48 hours in mosquitoes which had bitten a dengue patient upon the fourth day of the disease. Graham produced a very severe case of fever resembling dengue by inoculating a man subcutaneously with peptonized normal salt solution containing the salivary glands of a mosquito which had bitten a dengue patient 24 hours before. Graham's observations concerning the parasite in the blood and in the mosquito have not been confirmed, although the subject has been studied by several experienced microscopists. Carpenter and Sutton,[2] however, obtained two positive results out of four experimental cases of mosquito inoculation. The period of incubation in one of these, however, was two weeks, and the subjects were not sufficiently controlled to exclude the bites of other mosquitoes. Agramonte[3] studied an epidemic in Habana which was accompanied by a plague of *Culex fatigans*. He attempted to transmit the disease by mosquitoes, trying various species at various intervals after the insects had fed upon dengue patients, but did not succeed in producing the disease in this way. Guiteras and Finlay[4] endeavored to transmit the disease with *Culex pipiens*, but with negative results. Guiteras, Finlay, Agramonte, and others who have worked upon this subject state that their faith remains unshaken that the mosquito acts as the vector of dengue, despite the negative results of their experiments.

Ashburn and Craig[5] in 1907 studied the disease in Manila and showed that the virus is contained in the blood during the febrile stage.

[1] *Jour. Trop. Med.*, 1903, Vol. VI, p. 209.
[2] *Jour. A. M. A.*, 1905, XLIV.
[3] *New York Med. Jour.*, 1906, LXXXIV.
[4] *Rev. Méd. Trop.*, 1906, Vol. VII, p. 53.
[5] *Philippine Jour. of Sci.*, Vol. II, No. 2, Section B, May 1, 1907.

The intravenous inoculation of filtered dengue blood into healthy men is followed by a typical attack of the disease. The cause of the disease is, therefore, probably ultramicroscopic. They transmitted the infection by the mosquito, *Culex fatigans,* and concluded that this is probably the most common method of transmission. The period of incubation in the experimental cases averaged 3 days and 14 hours. They concluded from their studies that dengue is "not a contagious disease, and is infectious in the same manner as are yellow fever and malaria."

All our preventive measures are now based upon the supposition that dengue is a mosquito-borne infection. At times dengue appears to be one of the most contagious of all diseases, for it spreads like wildfire and spares practically no one in the community. An instance showing the non-contagiousness of dengue is given by Persons, U. S. N.: A squad of marines from the U. S. S. *Baltimore* were given shore leave at Cavite. Twenty of the 24 marines who had been ashore came down with the disease after returning to the ship, while there was a total absence of infection among those who had remained aboard. Observations made at the Naval Hospital at Canacao demonstrated that in the mosquito-free wards the disease did not spread, whereas when the hospital was located at Cavite it was noted that practically every case admitted became infected with dengue while under treatment for the original complaint (Stitt).

FILARIASIS

The filaria is a long, slender filiform threadworm with a curved or spiral tail. The adult worms live in the connective tissue, lymphatics, and body cavities. The embryos or larvæ are found in great numbers in the blood. In several species of which the life history is known mosquitoes act as the intermediate host. The most important filariæ of man are: (1) *Filaria bancroftii,* the larva of which is known as *Filaria nocturna,* appearing in the blood at night and occurring especially in Australia and the tropics; (2) *Filaria loa,* the larva of which is known as *Filaria diurna,* occurring in the blood by day and prevalent in West Africa and India; (3) the *Filaria perstans,* the larva of which is known as *Filaria perstans,* which persists in the blood both day and night, and occurs especially in West Africa and a number of other places. None of these young worms do any appreciable injury in the blood; of the adult worms, only one, namely, *Filaria bancrofti,* can be viewed as serious, while the second species, *Filaria loa,* is more or less troublesome. According to Manson, we are hardly justified at present in assuming that all the other species are entirely without effect upon their hosts. These parasites infect man throughout the tropical and subtropical belt. In the United States the infection, while not very prevalent, is endemic as far north as Charleston.

According to Manson, *Culex fatigans,* and according to James the *Anopheles nigerrinus,* are the intermediate hosts. When fed on the blood of a filarial-infested individual, it is found that the filarial larvæ soon escape from their shields in the thickened blood within the stomach of the mosquito. They pierce the stomach wall, enter the thoracic muscles of the insect, pass through a metamorphosis which takes from 16 to 20 days (longer or shorter, according to atmospheric temperature); they now quit the thorax and a few find their way to the abdomen; the vast majority, however, pass forward through the prothorax and neck, and, entering the head, coil themselves up close to the base of the proboscis and beneath the pharynx and under surface of the cephalic ganglia. This account is taken from Manson, to whose personal interest in this disease we are indebted for the advances in our knowledge of the entire subject of filariasis. The wonderful preparations of Low may be seen at the London School of Tropical Medicine, showing the *Filaria nocturna* in the head and proboscis of the mosquito ready to come out when the proboscis of the insect pierces its victim. The fact that the mosquito is the intermediate host in conveying the infection of Filaria rests upon these observations and not upon experiments which demonstrate the actual transference of the disease. Whether the worm may obtain an entrance by any other channel or medium would, according to Manson, be hard to prove and rash to deny. Our correct preventive measures are based upon the theory that this is an insect-borne disease, although other possible modes of transference must not be neglected. Prophylaxis, therefore, depends upon the suppression of the mosquito and the prevention of the infective mosquito-bite. As it is not definitely known how many species of mosquitoes convey the infection, the preventive measures must be along general lines; a combination of those described under malaria and yellow fever, as well as general sanitation and personal hygiene.

FLIES

The true flies have but two wings, that is, they belong to the order Diptera. They comprise an enormous number of species. Not only have the flies a superiority in point of numbers, but entomologists are concluding that they probably stand at the head of the insect system in point of evolution; that is, they are the most highly specialized of all insects. Contrary to popular opinion, flies are poor scavengers. Most flies prefer the sunshine, but species vary greatly in their habits and breeding places. However, surprisingly little is known of the life history and habits of most flies. The subject lacks attraction—especially the maggots or larval stage. The life history of the house fly in general was, down to 1873, mentioned in only three European works, and few

exact facts were given. Dr. A. S. Packard, then of Salem, Mass., studied the house fly and gave descriptions of all its stages, showing that the growth of a generation from the egg to the adult occupies from 10 to 14 days. In 1895 Howard further traced the life history and indicated that 120 eggs are laid by a single female, and that a generation is produced every 10 days at the summer temperatures of Washington. There may be, therefore, 12 generations in a summer. If each female lays only 120 eggs (1,000 have been noted), we have the possibility of countless millions coming from a single fly during a single season. Allowing 1,000 flies to the ounce, it has been estimated that the total product of a single fly in 40 days would equal 810 pounds, provided only one-half of them survived; hence, the logical time to begin fly suppression is in the early spring. Flies transmit disease in one of several ways. The biting flies, such as the tsetse flies, which transmit sleeping sickness, inoculate the trypanosome directly into the system by piercing the skin with their mouth parts. The common house fly does not bite. Biting flies, such as the *Stomoxys calcitrans*, abound in the United States in stables, houses, and also in nature. They have recently been implicated as go-betweens in poliomyelitis, and also in anthrax, relapsing fever, horse sickness (Pferdesterbe), and epithelioma of fowls. Other blood-sucking genera, such as *Tabanus, Chrysops, Hæmatobia,* etc., are of common occurrence, but are not known to carry any infection regularly, although they might readily do so.

FIG. 25.—HOUSE FLY (*Musca domestica*), SHOWING PROBOSCIS IN THE ACT OF EATING SUGAR.

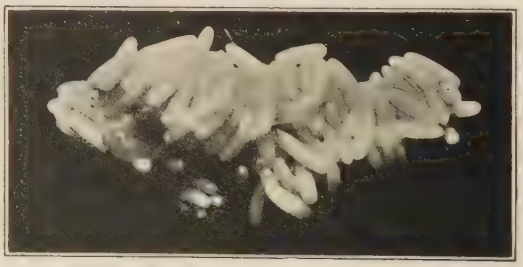

FIG. 26.—EGGS OF HOUSE FLY AS LAID IN A MASS.

The following brief account of the common house fly may be taken as a type of the life history and habits of flies in general. Remedies and preventive measures depend upon the peculiarities in the life history and habits of each particular genus and species.

Life History of the Musca Domestica.—A few adults live over the winter in cellars, barns, attics, and out-of-the-way places, and as soon

as warm weather sets in they lay their eggs in manure or organic refuse. In 6 to 8 hours the eggs hatch into larvæ (maggots), which grow rapidly and are fully developed in 4 or 5 days. Each larva then

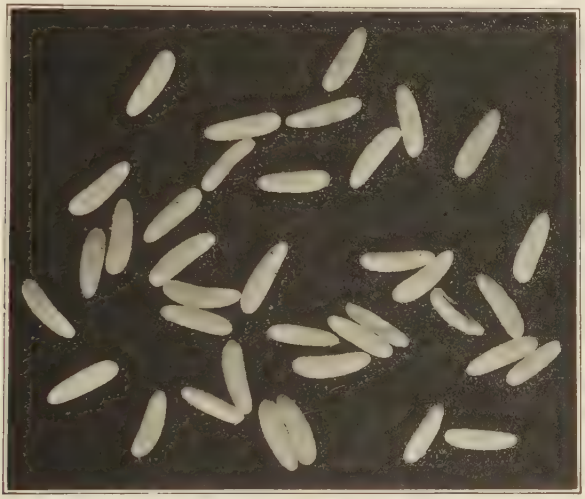

Fig. 27.—Eggs of House Fly. Some have hatched.

becomes a pupa in a hard brown case—the puparium. In 5 days more the pupal case opens and the adult fly appears for a season of activity covering several weeks. Most of them die in the early autumn, in great

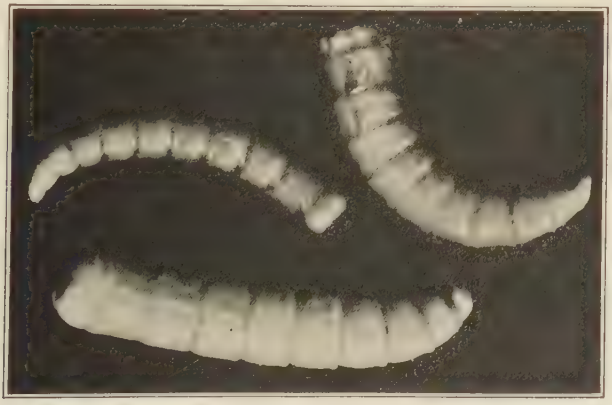

Fig. 28.—Larvæ of House Fly.

part due to a fungus disease, caused by *Empusa muscæ*, which becomes prevalent among the flies at this season of the year. A few are left and hibernate to continue the species. Hence, it takes about 10 days from egg to imago. It is, therefore, important to remove manure, garbage,

and other organic refuse at least as often as this in order to prevent the development of the winged insects.

The chief breeding place of common house flies is in horse manure. They also have been found to breed in human excrement, fermenting vegetable and putrefying animal matter, in the bedding in poultry pens, in refuse hog hair, in tallow vats, in carcasses of various animals, and in garbage and organic material of all kinds. All of which means that if we allow the accumulation of filth we will have house flies.

FIG. 29.—PUPARIUM OF HOUSE FLY.

Life History of Stomoxys Calcitrans.—*Stomoxys calcitrans,* the biting stable fly, is very similar to the house fly in its life history and in appearance during the preparatory stages, but develops more slowly, requiring nearly a month to undergo a complete life cycle. The eggs are laid like those of the house fly in horse manure, but more frequently in fermenting heaps of grass, cow-dung, brewer's refuse ("spent hops"), etc. The adult flies are much like the house fly, but have a sharp, needle-like proboscis. They feed exclusively on mammalian blood and are a great annoyance to horses and cattle in late summer and autumn. They bite persons less frequently, but are of importance on account of their relation to poliomyelitis, anthrax, etc. The stable fly can best be controlled by eliminating its breeding places.

FIG. 30.—STABLE FLY (*Stomoxys calcitrans*). (Brues.)

Flies as Mechanical Carriers of Infection.—Leidy in 1864 attributed the spread of gangrene in hospitals during the Civil War to the agency of the house fly. Shortly thereafter it was discovered that the bite of the gad-fly may transmit anthrax from cattle to man. Later it was found that purulent ophthalmia of the Egyptians is carried by the house

fly, and the spread of an infectious conjunctivitis known as "pink eye" in the South has been shown by Hubbard to be facilitated by little midges of the genus *Hippelates*. Reference has already been made to the bite of the tsetse flies in spreading nagana, sleeping sickness, and other trypanosomatic infections. Recently the stable fly has been shown to be able to transmit various infections in a mechanical way.

It is now known that typhoid fever and other intestinal infections may be transmitted by the common house fly. Celli in 1888 fed flies with pure cultures of typhoid, and showed that the virulent bacilli were passed in the dejecta. Kober in 1895 was one of the first to call special attention to the danger of contaminating food supplies by flies coming from the excreta of typhoid patients. The United States Army Commission—Reed, Vaughan, and Shakespeare—studied the presence of typhoid fever in our camps during the Spanish-American war in the summer of 1898. They concluded that flies undoubtedly serve as carriers of the infection. "Flies swarm over infected fecal matter in the pits and then deposit it and feed upon the food prepared for the soldiers at the mess tents. In some instances, where lime had recently been sprinkled over the contents of the pits, flies with their feet whitened with lime were seen walking over the food." Vaughan subsequently stated that he considered that about 15 per cent. of the cases of typhoid in the camps were caused by fly transmission.

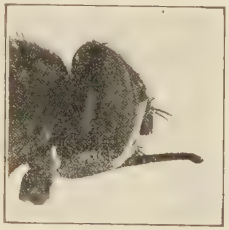

FIG. 31.—HEAD SHOWING PROBOSCIS, STOMOXYS CALCITRANS. (Brues.)

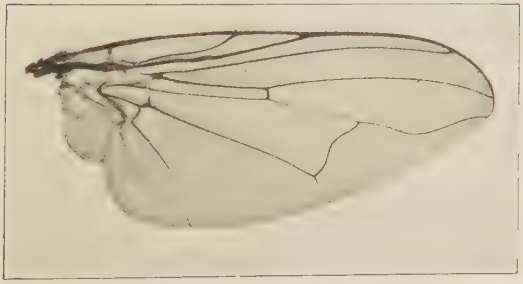

FIG. 32.—WING OF STABLE FLY (*Stomoxys calcitrans*).

Alice Hamilton[1] isolated typhoid bacilli from 5 out of 18 house flies captured in Chicago in the privies and fence near a sick room. It has been shown experimentally that living typhoid bacilli may remain in or upon the bodies of flies for as long as 23 days after infection.

Howard studied fly abundance in relation to the origin and prevalence of typhoid fever in the District of Columbia in the summer of 1908.[2] No particular correlation between the prevalence of the flies and the prevalence of the disease could be made out.

[1] *Jour. A. M. A.*, 1903, 40, p. 576.

[2] Rosenau, Lumsden, and Kastle: Report No. 3, 1908, P. H. and M. H. S., *Hygienic Laboratory Bull.* No. 52.

Flies undoubtedly spread the infection of typhoid fever, but the importance of the rôle they play in this regard varies considerably with circumstances. In camps, unsewered towns, and overcrowded places in poor sanitary condition the danger from flies may be considerable, but even under the worst conditions it is doubtful whether flies ever play the major rôle or are responsible for the bulk of typhoid fever, as has been stated. In a well-sewered city, such as Washington, we concluded that the flies are probably responsible but for an occasional case of the disease. It is very difficult in any particular instance to know quantitatively just how much of the infection is conveyed by flies and how much by contacts. The danger of flies is great enough without the need of exaggeration, and their suppression fully justifies the best energies of the health officer. It is perhaps a mistake to call the common house fly the "typhoid fly," not alone for the reason that the disease is spread in many other ways, but for the reason that the fly is responsible for the spread of many infections other than typhoid fever. Flies undoubtedly play the same rôle in dysentery, cholera, and all other intestinal infections that they do in typhoid fever. Tizzoni and Cattani in 1896 demonstrated active cholera organisms in the dejecta of flies caught in the cholera colonies of Bologna, Italy. These observations were subsequently verified and extended by Simonds, Offelman, McRae, and others.

Fig. 33.—The "Little House Fly" (*Homalomyia canicularis* ♂). (Hewitt.)

It is now quite evident that flies lighting upon a case of smallpox, measles, scarlet fever, and other exanthematous disease may very readily transmit these infections to another person. I have actually seen maggots breeding in the open lesions of a case of smallpox treated in the open air at Eagle Pass, Texas.

Flies may, in the same mechanical way, transmit the infection of erysipelas, anthrax, glanders, and other skin infections. It is known that flies may ingest tuberculous sputum and excrete tubercle bacilli which may remain virulent as long as 15 days. Flies have also been associated with leprosy and many other diseases.

Esten and Mason[1] counted the bacterial population of 415 flies and found that the number of bacteria on a single fly may range all the

[1] *Store's Agricultural Experiment Station, Bull. No. 51,* April, 1908.

way from 550 to 6,600,000. Early in the fly season the numbers of bacteria on flies are comparatively small, while later the numbers are comparatively very large. The places where flies live also determine largely the number of bacteria they carry. The average of the 415 flies was about one and one-quarter million bacteria. The method of the experiment was to introduce the flies into a sterile bottle and pour into the bottle a known quantity of sterilized water, then shake the bottle to wash the bacteria from the body of the fly. The numbers, therefore, only represent those carried on the outside and not those in the intestinal tract. The experiments of Esten and Mason were designed to simulate the number of microorganisms that would come from a fly in falling into milk.

Torrey[1] found that a single fly may carry from 570 to 4,400,000 bacteria upon its surface, and from 16,000 to 28,000,000 in its intestinal tract. The prevailing types are *Streptococcus equinus fecalis* and

Fig. 34.—Wing of House Fly, Showing How It Carries Dust Particles.

salivarius, which are also found in the breeding and feeding places of the house fly. Torrey also obtained three cultures of *B. paratyphosus,* which is especially significant.

Even though flies breed in manure, and the larvæ teem with bacteria, the adult winged insect, when newly hatched, contains fewer microorganisms. This cleansing is due to the active phagocytosis which takes place during metamorphosis from pupa to imago. The bacteria in the intestinal tract of the newly hatched imago are mostly extruded soon after emergence from the puparium.

Bacot,[2] however, has shown that certain species of bacilli ingested during the larval period of *Musca domestica* can retain their existence while their host is undergoing the process of metamorphosis, and continue their existence in the gut of the adult fly, but that their number diminishes suddenly after emergence. In a subsequent work Bacot[3]

[1] *J. A. M. A.,* May 11, 1912, LVIII, No. 19, p. 1445.
[2] *Trans. Ento. Soc.,* London, 1911, Part II, p. 497.
[3] *Parasitology,* IV, I, Mar., 1911, p. 68.

demonstrated that *Bacillus pyocyaneus* may thus survive. Faichnie[1] shows how *B. typhosus* may also persist. Ledingham confirms these conclusions, and states that he has recently isolated *B. typhosus* from pupa, the larvæ of which have fed on this organism.

Graham-Smith[2] recovered *B. anthracis* from blow flies bred from larvæ fed on meat infected with the organism, but failed to recover *B. typhosus* and *B. enteritidis*.

Among the list of diseases of which there is more or less evidence that the infection may be conveyed by flies are: typhoid, cholera, dysentery, diarrhea in infants, anthrax, yaws, erysipelas, ophthalmia, diphtheria, smallpox, plague, tropical sore, parasitic worms, sleeping sickness, poliomyelitis, relapsing fever, and several infections of the lower animals.

An interesting light was thrown on the possible modes of dissemination of the eggs and larvæ of hookworms by Galli-Valerio (1905). He placed eggs and larvæ of *Ankylostoma duodenalis* in a bottle with flies, and on washing found many eggs and encapsulated larvæ which had adhered to their bodies, but none in the flies' intestines.

Flies may transmit the virus of disease mechanically, either through their dejecta or upon their mouth parts, legs, and other surfaces of the body. The flies may carry the infection directly to our lips or indirectly to our food or to any surface upon which they light.

Suppression.—The suppression of the common house fly may be accomplished by striking at their breeding places. In a city this does not present very great difficulty. It resolves itself simply into a matter of cleanliness—organic cleanliness of our environment. The chief breeding places are in horse manure and garbage. These should be given first attention. One neglected stable will furnish a plague of flies for an entire neighborhood. Their suppression in a well ordered city fortunately is neither expensive nor difficult, but it requires a well-trained and capable corps of inspectors with sufficient authority to enforce the regulations. The suppression of flies by voluntary effort through the slow process of education cannot be relied upon.

In cities stable manure should be placed in properly covered receptacles and removed at least once a week. This one measure obviates the use of kerosene, chlorid of lime, Paris green, or arsenate of lead, all of which are expensive and uncertain unless used frequently and in liberal amounts; further, they decrease the fertilizing value of the manure.

Garbage should be kept in water-tight cans with good covers and removed frequently, especially in the warm weather. Refuse on city lots, in back yards, in alleys, about wharves, markets, and similar places where organic matter collects should be regularly and faithfully taken

[1] *Jour. Roy. Army Med. Corps*, XIII, 1909.
[2] Repts. to Local Gov. Bd., New Series, No. 53, 1911.

away. Householders, provision merchants, storekeepers, and others should be held responsible for the cleanliness and tidiness of their premises, and those who violate these simple and primitive hygienic requirements should have their places cleaned up for them at their own expense. The orders of the Health Department of the District of Columbia, published May 3, 1906, are excellent, and, if carried out, would be very effective. These orders may be briefly condensed as follows:

FIG. 35.—THE HODGE FLY TRAP ON A GARBAGE CAN.

All stalls in which animals are kept shall have the surface of the ground covered with a water-tight floor. Every person occupying a building where domestic animals are kept shall maintain, in connection therewith, a bin or pit for the reception of manure, and, pending the removal from the premises of the manure from the animal or animals, shall place such manure in said bin or pit. This bin shall be so constructed as to exclude rain water, and shall in all other respects be water-tight, except as it may be connected with the public sewer. It shall be provided with a suitable cover and constructed so as to prevent the ingress and egress of flies. No person owning a stable shall keep any manure or permit any manure to be kept in or upon any portion of the premises other than the bin or pit described, nor shall he allow any such bin or pit to be overfilled or needlessly uncovered. Horse manure may be kept tightly rammed into well-covered barrels for the purpose of removal in such barrels. Every person keeping manure in any of the more densely populated parts of the District shall cause all such manure to be removed from the premises at least twice every week between June 1 and October 31, and at least once every week between November 1 and May 31. No person shall remove or transport any manure over any public highway in any of the more densely populated parts of the District except in a tight vehicle, which, if not closed, must be effectually covered with canvas, so as to prevent the manure from being dropped. No person shall deposit manure removed from the bins or pits within any of the more densely populated parts of the District without a permit from the health officer. Any person violating any of these provisions shall, upon conviction thereof, be punished by a fine of not more than $40 for each offense.

In addition to this ordinance, others have been issued by the health department of the District of Columbia which provide against the contamination of exposed food by flies and by dust. The ordinances are excellently worded so as to cover all possible cases. They provide for the registration of all stores, markets, cafés, lunch rooms, or any other places where food or beverage is manufactured, prepared, stored, offered for sale, or sold.

Where it is not practicable to remove manure, it may be kept covered in a dark place, which discourages the visitation and breeding of flies, and in addition should be carefully screened. Flies may be destroyed with sulphur dioxid, carbon bisulphid, hydrocyanic acid gas, petroleum, chlorinated lime, Paris green, and other insecticides. Kerosene (petroleum) poured upon manure, garbage, and other fly-breeding places is effective; it kills the larvæ. Lime is not effective; chlorinated lime is good, but is not practical, for, like all other substances used for this purpose, it needs frequent application and in generous amounts.

Flies are thirsty insects and will be attracted to a saucer of water containing a little formalin (4 per cent.). This simple measure will kill many of them in a room. The salts of barium, cobalt, and other poisons, such as arsenic, potassium bichromate, or quassia infusion, may be used instead of formalin, and are better bait if sweetened. Sticky fly-paper, fly traps, electric fans, and other well-known measures will help dispose of a certain number of flies, but all these measures are tentative, and attack the problem at the wrong end.

The fly has a number of natural enemies: various fungi, especially one belonging to the *Entomophthoreæ*, which destroys flies in the autumn. Flies also harbor protozoa and nematodes as parasites, which, however, seem to do them little harm. The little bright red objects often seen attached to flies are mites, which are usually only temporary ectoparasites stealing a free ride. When spider webs are not disturbed they catch, and the spiders devour, a large number of flies. The house centipede (*Scutigera*) also sometimes catches and eats flies, as do the common garden toad, some lizards, and a few insectivorous birds.

Flies and similar dipterous insects are responsible for the transmission of a large number of diseases, most of which are discussed elsewhere. It now remains to consider sleeping sickness, transmitted by the tsetse fly (*Glossina palpalis*), and pappataci fever, transmitted by a biting dipterous insect (*Phlebotomus pappatasii*). For convenience a general consideration of the trypanosomes is inserted in this chapter.

SLEEPING SICKNESS

Sleeping sickness was limited to tropical Africa, especially in the Congo, on the shores of Victoria Nyanza, and about the head waters

of the Nile, but is gradually spreading. Many thousands have perished from this infection, caused by *Trypanosoma gambiense* and transmitted by the tsetse fly (*Glossina palpalis*). The disease is characterized by two stages: in the first there are irregular fever, glandular enlargements, an erythematous rash, and localized edemas. In the second there are slowly increasing lethargy and other morbid, nervous symptoms. After a chronic course sleeping sickness usually terminates in death; few cases recover. Many instances of fatal homesickness in the negroes during the slave trade are now believed to have been this disease.

The *Trypanosoma gambiense* was discovered by Dutton in 1901 during the first or febrile stage of sleeping sickness, and subsequently studied by Dutton and Todd, who did not at first suspect the relation of the trypanosome to sleeping sickness. This was shown by Castellane in 1903. The trypanosomes are found in the cerebrospinal fluid, in the enlarged lymphatic glands, and also in the circulating blood. It seems that when the trypanosomes are inoculated through the skin by the tsetse fly they are temporarily blocked by the lymphatic glands. From here small numbers of them pass into the circulation and thus to other parts of the body. They are always in the fluids; never in the cells or tissues. Novy and McNeal in 1903 accomplished the remarkable feat of growing trypanosomes in the water of condensation of blood agar tubes. Pure cultures show marked differences between the *Trypanosoma lewisi* of the rat and the *Trypanosoma grussei* of horses and other domestic animals. So far no one has succeeded in cultivating the *Trypanosoma gambiense* in artificial culture media.

Fig. 36.—Tsetse Fly (*Glossina palpalis*).

The relation of the tsetse fly to the transmission of this disease rests upon satisfactory evidence. Dutton and Todd, as well as others, find these flies abundant wherever sleeping sickness exists. Wherever the *Glossina palpalis* is absent sleeping sickness never spreads, as Koch observed; while, on the other hand, if a case is brought to a locality where the tsetse fly prevails, it soon spreads. It is probable that the transmission by the tsetse fly is not of the simple mechanical type, but that the parasite undergoes a sexual evolution within the insect. Flies seem to lose their power of

transmission soon after feeding on an infected animal, and Bruce considers it thoroughly impossible that mechanical transmission alone could explain the situation. Kleine's experiment on monkeys, confirmed by Bruce, showed that the flies may convey the disease 21 days after one feeding upon a monkey infected with sleeping sickness. In another experiment by Taute,. which is reported by Kleine, infection was produced on each of the first three days after feeding. From the fourth to the tenth day no infection resulted. The flies then became infective again and produced the disease from the eleventh to the forty-fourth day. Kleine[1] concludes that the period of development or intrinsic period of incubation in the fly is about 20 days or a little less. Flies remain infective at least 75 days. Not all flies which drink blood containing trypanosomes become infective. The proportion is about 1 in 20. Of the flies caught in nature in endemic areas, from 2 to 10 in one thousand are capable of transmitting the disease to animals. Novy has emphasized, and Minchim has corroborated the fact, that tsetse flies may harbor non-pathogenic as well as pathogenic trypanosomes, a fact which impairs the value of a great deal of the microscopic work which has been done. As a means of avoiding the accident of dealing with naturally infected flies, it is best to use those which have been bred and raised in the laboratory.

Prevention.—The prevention of sleeping sickness in the present state of our knowledge depends first upon isolation of the sick, protecting both the sick and the well against fly bites, and the suppression of the flies themselves. The sick should be isolated in a location where *Glossina palpalis* is absent, or in a well-screened and carefully managed hospital. It is especially important to isolate all those who carry the infection in the early stages of the disease, whether they feel sick or not. It is not sufficient simply to isolate those who have enlarged glands, but careful blood examinations must be made. The trypanosomes have been found in the circulating blood of persons with normal lymph glands.

All persons taken to the hospital and detention station are given a thorough treatment with atoxyl (a combination of arsenious acid and anilin oil). Atoxyl is one-tenth as toxic and contains about three times as much arsenic as arsenious acid alone. The dose is from ¾ to 3 grains (0.05-0.2 grams) subcutaneously.

The extermination of the tsetse fly seems a hopeless task. The larvæ remain in the body of the mother fly until fully developed and are then dropped on moist soil, in which they burrow to undergo transformation to the adult state; therefore, clearing of the land in limited locations largely diminishes the number of flies. Clearing the brush exposes the earth to the sun, and the surface becomes dry and hard, so

[1] *Bull. of the Sleeping Sickness Bureau*, No. 7, 1909.

that flies die during the pupal period. This measure has limited possibilities, but is useful, as Shirata points out, around ports, in the neighborhood of villages, wharves, and other places.

The tsetse fly may also be fought by suppressing its food supply. It must obtain the blood of some vertebrate animal every two or three days.

FIG. 37.—VARIOUS GNATS

1. Culicoides milnei, Austen.
2. Culicoides brucei, Austen.
3. Culicoides grahamii, Austen.
4. Phlebotomus duboscqi, Neven-Lemaire
5. Simulium latipes, Mg.
6. Simulium damnosum, Theob.
7. Simulium wellmanni, Rouband.
8. Simulium griseicollis, Becker.

Simulium is implicated in pellagra; Phlebotomus in pappataci fever.

The German Commission has shown that on the banks of the Victoria Nyanza the tsetse fly lives largely upon crocodile blood. This fact was discovered by the interesting observation that the flies frequently contain parasites peculiar to the crocodile's blood. Koch believes that the disease may be successfully controlled by destruction of the crocodiles, a theory which later research has rendered very unlikely.

THE IMPORTANT TRYPANOSOMES—(Modified from Nacht and Mayer)

Infection According to R. Koch		Zoological Name	First Described by	Name of the Disease	Animals Infected in Nature	Geographical Distribution	Transmitted by
Group I: Constant in morphology, virulence and host.		*Tr. lewisi* (Kent, 1880)	Chaussat, 1850 Lewis, 1878	Generally produces no noticeable symptoms	Rats, *Mus rattus decumanus, rudescens*	Apparently everywhere these rats occur	Rat fleas (according to Rabinowitsch and Kempner) *Hæmatopinus spinulosus* (Burmeister) according to v. Prowazek
	Group II: Inconstant in morphology, virulence and host.	*Tr. theileri* (Laveran, Bruce, 1902) *Tr. evansi* (Steel, 1885)	Theiler, 1902 Evans, 1880	Gall sickness Surra	Cattle Horses, cattle, camels, dogs and other mammals	South Africa (Transvaal, Orange Cape), India, Indo China, Philippines, Mauritius, North Africa	*Hippobosca rufipes* (?) *Tabanus tropicus* and *lineola* (?) *Stomoxys calcitrans* and *nigra* (?)
		Tr. brucei (Plimmer & Bradfort, 1889)	Bruce, 1894	Nagana	Most mammals, especially domestic animals	Greater part of Africa	*Glossina morsitans* and *fusca* *Glossina pallidipes* (?)
		Tr. equiperdum (Doflein, 1901)	Rouget, 1894 Schneider & Buffard, 1899	Dourine	Horses	Europe (Spain, Hungary, South Russia), south coast of Mediterranean, North Africa, Asia Minor, Persia, N. America, Chile (?)	Transmitted by coitus
		Tr. equinum (Voges, 1901)	Elmassian, 1901	Mal de Cadéras	Horses	South America (Argentine, Bolivia, Brazil, Uruguay, Paraguay)	*Mosca brava*: *Stomoxys calcitrans* and *nebulosa* (?)
		Tr. gambiense (Dutton, 1902) *Tr. Castellani* (Kruse, 1903) *S. ugandense* (Castellani, 1903)	Dutton, 1901 Castellani, 1903	Sleeping sickness	Man	Equatorial Africa	*Glossina palpalis*
		Tr. dimorphon (Dutton & Todd, 1904)	Dutton and Todd, 1904		Horses	Senegambia	*Glossina palpalis* (?) *Stomoxys* (?)
		Schizotrypanum cruzi	Aragao, 1910	Barbiero fever	Man	South America	Conorhinus megistus

Todd and Wolbach [1] suggest a systematic examination of the natives in the endemic area by gland palpation and gland puncture. The latter consists in withdrawing a drop of fluid from one of the enlarged lymphatic glands by means of a hypodermic syringe. The little drop of bloody fluid thus obtained is examined as a fresh preparation under the microscope for trypanosomes. By this method these investigators found at least 0.8 per cent. of the population of the Gambia to harbor trypanosomes. If all the infected individuals could be collected in villages for observation, treatment, and isolation, it would do much to limit the disease.

Trypanosomes are the cause of numerous other diseases in animals, as will be seen by reference to the table on page 236. So far as known, sleeping sickness is the only important disease of man produced by trypanosomes. Kala-azar, however, is produced by a flagellated protozoon parasite which probably belongs to the trypanosomes.

Practically all animals are susceptible to almost all trypanosomes. The trypanosomes which infect man may readily be transmitted to monkeys, guinea-pigs, rabbits, etc.

PAPPATACI FEVER

Doerr and Russ [2] and also Doerr, Franz, and Taussig originally described a three-day fever which occurs on the shores of the Adriatic, the cause of which is not known, but which is of special interest for the reason that it has been demonstrated to be transmitted through the bite of a dipterous insect commonly called a gnat—*Phlebotomus pappatasii.*

FLEAS

Fleas are flat, wingless insects related to the Diptera. They pass through a complete metamorphosis: embryo, larva, pupa, and imago. The adult female flea deposits her eggs among the hair or fur of the host animal, but, unlike the eggs of many ectoparasites, they are not fastened to the hairs and therefore fall freely to the ground. The eggs are oval, whitish, and smooth and about half a millimeter long. The larvæ escape from the eggs in 2 to 5 days. They are able to break the egg shell by a slender process on the top of the head which disappears after the first molt. The larva is a slender, legless, cylindrical creature, whitish or yellowish in color, with a head and 13 segments. There are a few scattered hairs or bristles on the body, and at the tip is a pair of corneus processes. At the front of the head is a pair of biting jaws or

[1] *Annals of Tropical Medicine and Parasitology,* Vol. V, No. 2, Aug., 1911, p. 245.

[2] *Schiffs und Tropen Hyg.,* 1909, Vol. XIII, No. 22, p. 693.

mandibles. The larvæ feed on almost any kind of refuse. They have been reared on the sweepings from rooms. There is always some organic matter in such dust, and this is doubtless their nourishment. In houses the larvæ usually crawl into cracks or in carpets, where they feed

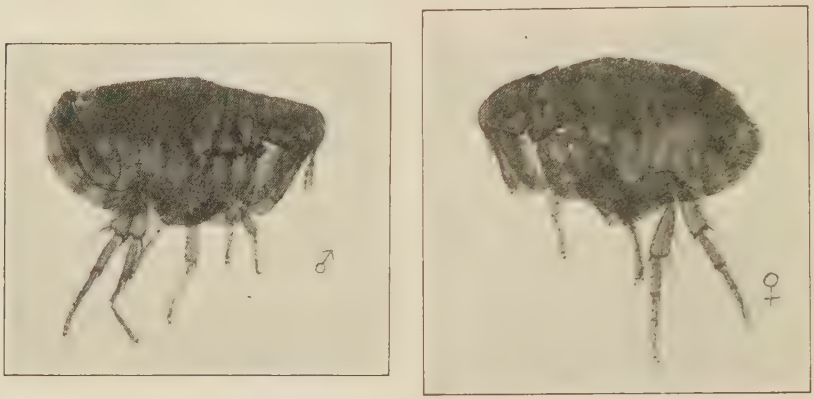

Fig. 38.—The Indian Rat Flea (*Læmopsylla cheopis Rothsc.*).

and grow. Those that infest wild animals probably feed on the refuse in the nests or retreats of these animals. It will be noticed that, contrary to the mosquito, the larval and pupal stages of the flea are not aquatic. They remain in the larval stage from a week to ten days, sometimes two weeks, molting the skin three times in this interval. Then they spin flat, white, silken cocoons in which they transform to the pupal stage. In from 5 to 8 days the adult flea emerges from the cocoon. The period of their transformation is affected by the temperature and moisture. In warm, damp weather a generation may develop in ten days or two weeks, but usually about 18 days to three weeks elapse from the egg to the adult. Although some moisture is necessary for their development, an excess is apt to destroy the larvæ.

The leaping ability of adult fleas is familiar to all. This, however, has been greatly exaggerated. The British Plague Commission determined that fleas jump 3 to 5 inches, never over 6. No part of the leg is particularly enlarged, so that the jump is made by the entire leg, as in the leaf-hopper insect, and not by the femur of the hind leg, as in the grass-hopper. Fleas do not vary much in size. They are mostly about 2 to 3 millimeters long. The adult insect has a hard, strongly chitinized body. The mouth parts resemble somewhat those of the mosquito. Both the male and the female flea are capable of piercing the skin to obtain blood and thus transmit infection. Fleas, as a rule, prefer certain hosts, but are not as particular in this regard as are many parasites. Those species which are best known are found to attack several hosts, including man. This is one reason that makes

them dangerous parasites, so far as plague and other infections are concerned. Over 300 species are described. Formerly all fleas were classified in the single family Pulicidæ, genus *Pulex;* now they are arranged in many genera and these genera grouped into families.[1]

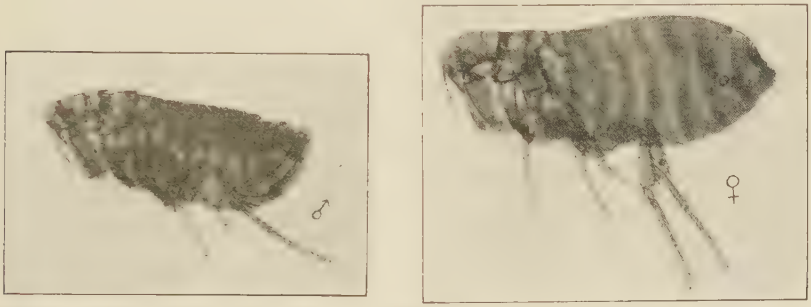

Fig. 39.—The Common Rat Flea of Europe and North America (*Ceratophyllus fasciatus Bosc.*).

Pulex serraticeps or *Ctenocephalus canis* occurs all over the world, infesting cats and dogs, also many other animals. They are frequently brought into houses upon domestic animals, and thus become troublesome to man. *Pulex irritans* is the human flea, sometimes called the "house flea" or "common flea."

Fig. 40.—The Human Flea (*Pulex irritans Linn.*).

The fleas concerned in the transmission of plague are *Læmopsylla cheopis,* the Indian rat flea, and *Ceratophyllus fasciatus,* the common rat flea of Europe and North America. Plague may also be transmitted by *Ctenocephalus felis,* the cat flea; *Pulex irritans,* the human flea; *Ceratophyllus acutus,* the squirrel flea, and doubtless other genera and species.

[1] Banks: "The Rat and Its Relation to the Public Health," P. H. and M. H. S., p. 69.

In addition fleas act as intermediate hosts for certain tapeworms (*Dipylidium caninum*), and doubtless are the mechanical or biological carriers of other infections. Nicolle incriminates the flea in typhus fever.

Pulicides.—Adult fleas succumb to the agents applicable to insects in general. Mitzmain [1] has shown that water is of little value in the destruction of mature fleas. Glycerin is also practically inert as a pulicide, but tincture of green soap is very quick and effective. This action cannot be due to the alcohol in the soap, for alcohol in the strength of 70 per cent. and absolute is uncertain in its action and practically inefficient. Kerosene (coal oil) is a very efficient flea destroyer. Formalin, phenol, mercuric bichlorid, and tricresol in the strength used as disinfectants are of little value in killing fleas. Powdered sulphur seems to be of no value.

Of gases, bisulphid of carbon (CS_2), hydrocyanic acid gas (HCN), and sulphur dioxid (SO_2) are highly efficient in the strengths recommended for general insecticidal purposes. Chloroform or ether first anesthetizes fleas, and if continued kills them. This is important for the safe handling of rats, squirrels, and other plague animals. The host may be chloroformed and the fleas and other ectoparasites removed with a comb. The anesthetic may be controlled by practice so that the host will recover and the fleas die, or both recover, or both die, as may be desired.

In flea-infected houses the larvæ, living in the cracks of the floor, etc., may be easily controlled by sprinkling a thin coating of flake naphthalene on the floor and then leaving the room tightly closed over night. In the morning the naphthalene may be swept up and what remains used again.

RELATION OF PLAGUE TO RATS AND FLEAS

Plague is primarily a disease of the rat and secondarily of man. This fact is now firmly established not only by the recent experiences, but especially through the admirable studies of the Indian Plague Commission,[2] which established beyond doubt the fact that plague may be and generally is transmitted from rat to rat and from rat to man through the agency of the flea—*Lœmopsylla cheopis*—and sometimes by *Ceratophyllus fasciatus, et al.* During some plague epidemics it has been noted that the rats die in great numbers before and during the outbreak. It is now known that this epizootic in the rat is true plague. In nature, rats suffer both with acute and chronic plague.

In the laboratory, rats may be infected with plague by ingestion, by

[1] *Public Health Reports*, July 29, 1910, Vol. XXV, No. 30, p. 1039.
[2] *Journal of Hygiene*, Vol. VI, No. 4; Vol. VII, Nos. 3, 6; Vol. VIII, No. 2.

application of the virus to mucous or cutaneous surfaces, or by subcutaneous inoculation. In nature, rats may become infected by any of these means or through flea bites.

Rats are great travelers, and have carried the plague to all quarters of the globe. A more complete discussion of the rat and its relation to plague and other diseases will be found on page 242.

Within the past few years it has been discovered that, while the rat is the great medium for the spread of plague, the disease is probably preserved from extinction in Thibet by another rodent, the marmot (*Arctomys bobac*). In California the infection has gotten into the ground squirrels (*Citellus beecheyi*), in which the disease will doubtless be kept alive for many years to come. To realize the full importance of these discoveries, it is only necessary to call to mind that, in order to eradicate plague forever from the surface of the globe, a war-

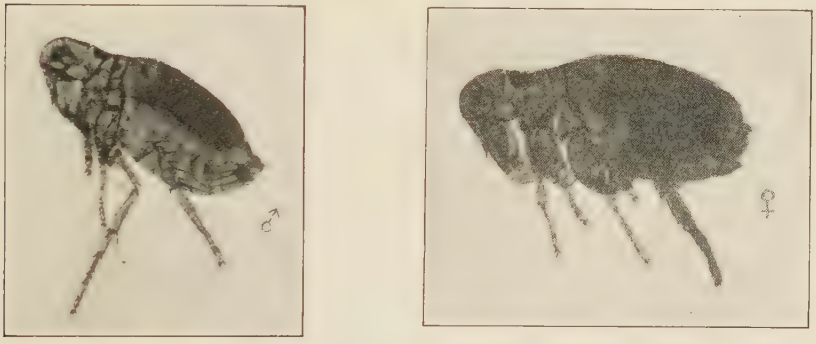

FIG. 41.—A SQUIRREL FLEA (*Hoplopsyllus anomalus Baker.*).

fare against the rat alone is not sufficient, but must include the rodents mentioned and perhaps others.

Simond in 1897 advanced the theory that plague was carried by fleas. This theory was developed by J. Ashburton Thompson and others and conclusively proved by the Indian Plague Commission. The exact method by which the flea transmits the infection from animal to animal is not definitely understood. The mouth parts appear not to remain infected. It is possible that the salivary secretions contain the microorganisms. It is known that the plague bacilli may live in the digestive tract and be passed in live and virulent numbers in the dejecta. It is easy to understand how some of the infected dejecta may be rubbed or scratched into the little wound produced by the flea bite. When it was found that the common rat flea of Europe, the *Ceratophyllus fasciatus,* does not readily bite man, considerable doubt was thrown upon the part played by the flea in plague transmission. These negative results, however, are offset by the convincing positive proofs of the British Plague Commission in India, and by McCoy and Mitzmain in

San Francisco, who showed that under certain conditions the rat flea will bite man, especially if the natural food supply is limited, and that these fleas may feed on a man's hand even in the presence of a rat.

Raybaud [1] calls attention to the fact that the rat flea (*Ceratophyllus fasciatus*) is able to hibernate for a month or 45 days without nourishment, and that virulent plague germs may persist unharmed in its stomach during this length of time and even longer. This fact may be of importance for the transmission of plague to a distance.

It should be remembered that, according to the observations of Nuttall and Yersin, flies and possibly other insects may also occasionally convey the infection. Walker [2] considers, as the result of experiments, that bedbugs and other biting insects play an important rôle in the transmission of plague.

RATS AND OTHER RODENTS

Rats, mice, squirrels, and other rodents have become a serious problem in preventive medicine, and their habits and methods of suppression may be considered conveniently at this place. Plague being primarily a disease of rats, the prevention and suppression of this infection resolve themselves into a war upon these rodents. For the control of plague it is, therefore, necessary to have a knowledge of the life history and methods of attacking the problem in the lower animals. In addition to plague, rats are the great reservoir of trichinosis. They are responsible for the transmission of certain tapeworms and other parasites. They are subject to leprosy, cancer, and numerous other diseases, some of which concern man.

Rodents comprise more than one-third of all living species of mammals, and exceed any other mammalian order in the number of individuals. They have no canine teeth, but strongly developed incisors. Only the front of the incisors is covered with enamel, which keeps them sharp and chisel-like, owing to the more rapid wearing away of the softer dentine. The incisor teeth continue to grow throughout the life of the animal. The most extensive family of rodents is the *Muridæ*, which includes the true rats and mice, typified by the genus *Mus*. Trouessart, in his "Catalogus mammalium," enumerates 250 species of *Mus* described before 1905. Since that date a number of new forms have been described.

The genus *Mus* is characterized by narrow, ungrooved incisors; three small-rooted molars; soft fur mixed with hairs, sometimes with spines; a rudimentary pollex (thumb) having a short nail instead of a claw; a long tail bearing rings or overlapping scales and often naked or

[1] *Presse Médicale*, March 8, 1911, No. 20.
[2] Walker: *Indian Med. Gaz.*, 1910, No. 3, p. 93.

nearly so. The ears are rather large, the eyes bright and prominent, and the muzzle somewhat pointed.

The distinction between rats and mice is arbitrary and based on size. Of the many species of the genus *Mus* only three or four have developed the ability to adapt themselves to such a variety of conditions as to become cosmopolitan. Four have found lodgment in America:

The common house mouse, *Mus musculus.*
The English black rat, *Mus rattus.*
The Egyptian or roof rat, *Mus alexandrinus.*
The brown rat, *Mus norvegicus.*

The black rat and the roof rat differ from each other mostly in color, and some zoölogists regard them as races of the same species. The brown rat is also known as the gray rat, barn rat, wharf rat, sewer rat, and Norway rat.

The black rat (*Mus rattus*) has been known in Europe since the twelfth century, and from there has been carried to America. The brown rat (*Mus norvegicus*) came later, and, as it is more destructive, larger, and more ferocious, it is rapidly driving the black rat before it. The brown rat differs somewhat in habits from the black rat, especially in that it burrows, which protects it against its enemies and renders its suppression more difficult.

The house mouse holds its own everywhere against the brown or Norway rat, as it is able to get into holes too small for the rat to follow. Albinism and melinism occur in all species; pied forms are common. The white rat of the laboratory is an albino form of either *Mus rattus* or *Mus norvegicus.*

Breeding and Prevalence.—The brown rat is more prolific than either the roof rat or the black rat. The brown rat reproduces from three to five times a year, each time bringing forth from six to nine, and sometimes as many as 22 or 23, young. They breed more rapidly in temperate and equable climates than in those of great variability. The number of rats is only limited by the food supply and opportunities to nest. Few people have any conception of the enormous numbers of rats in cities and on farms. Although few are seen in the day time, at night they fairly swarm along river fronts and wharves, as well as in sewers, stables, warehouses, markets, and other places where food may be found. A few instances will illustrate the prolific habits and give an idea of the destructive tendency of rats.

In 1901 an estate near Chichester, England, was badly infested with rats;[1] 31,981 were killed by traps, poisons, and ferrets, while it is esti-

[1] *The Field,* London, Vol. C, p. 545, 1902.

mated that tenants, at the threshing, destroyed fully 5,000 more. Even then the property was by no means free from rats.

During the plague of rats on the island of Jamaica, in 1833, the number killed on a single plantation in a year was 38,000.[1] The injury to sugar cane on the island caused by the animals was at that time estimated at half a million dollars a year.

The report of the Indian Famine Commission in 1881 affords one of the best illustrations of the number of rats that may infest a country. An extraordinary number of the animals at that time inhabited the Southern Deccan and Mahratta districts of India.[2] The autumn crop of 1878 and the spring crop of 1879 were both below the average, and a large portion of each was destroyed by rats. The resulting scarcity of food led to the payment of rewards for the destruction of the pests, and over 12,000,000 were killed.

Migration.—The migrations of rats have often been recorded. The brown rat is known in Europe quite generally as the migratory rat; the Germans call it the Wanderratte. Pallas relates that in the autumn of 1772 they arrived from the East at Astrakhan, southeastern Russia, in such great numbers and so suddenly that nothing could be done to oppose them. They crossed the Volga in immense troops. The cause of this general migration was attributed to an earthquake, but, since similar movements of the same species often occur without earthquakes, it is probable that only the food supply of the animals was involved in the migration which first brought the brown rat to Europe.

Seasonal movements of rats from houses and barns to the open fields take place in the spring, when green and succulent plant food is ready for them. The return movement takes place in the autumn. This seasonal migration is notable even in large cities. In 1903 a multitude of migrating rats spread over several counties of western Illinois. They traveled in great armies and invaded the farms and villages of Rock Island and Mercer counties, and caused heavy losses during the winter and summer of 1904. In one month Mr. Montgomery of Mercer county killed 3,435 rats on his farm. He caught most of them in traps.

In England a general movement of rats inland from the coast occurs every October. This is known to be closely connected with the closing of the herring season. During the fishing the rodents swarm to the coast attracted by the offal left in cleaning the herring, and when this food fails the animals troop back to the farms and villages.

An invasion of rats (*Mus rattus*) in the Bermuda Islands occurred about the year 1615. Within two years they had increased so alarmingly that none of the islands was free from them. The rodents "devoured

[1] *New England Farmer*, Vol. XII, p. 315, 1834.
[2] *British Med. Jour.*, Sept. 16, 1905, p. 623.

everything that came in their way—fruits, plants, and even trees"—so that for a year or two the people were nearly destitute of food. A law was passed requiring every man in the island to keep 12 traps. In spite of all efforts the animals continued to increase until they finally disappeared, so suddenly that it is supposed they must have been victims of a pestilence.

While stationed upon Angel Island in San Francisco harbor I observed several migrations of rats between the army post and the quarantine station, which were about a mile apart and separated by an intervening ridge. Everyone is familiar with the sudden invasion of stores, factories, and other structures with these rodent pests, which causes considerable economic loss.

On Vessels.—Rats are found on all vessels; they are great travelers. It is through this seagoing tendency that the rat has become cosmopolitan. Rats get on board vessels readily as they lie at their dock; sometimes they are carried on board in the cargo.

It is very important to prevent the introduction of rats on vessels at plague-infected ports; it is also important to prevent the passage of rats from ship to shore, particularly if the vessel is from a plague port. In order to accomplish this, it is necessary to exercise particular care. In extreme cases the ship should not approach the dock, but the cargo should be handled by means of lighters. When the ship lies at its moorings in a stream or in the open bay rats may get on board by swimming, and climbing in through the hawse pipe. Rats rarely swim more than one-quarter to one-third of a mile. If the vessel ties up at the dock, inverted funnels should be placed on the hawsers. The gangplanks should be watched during the day and always taken up at night. Vessels from plague ports should always be treated with sulphur dioxid, preferably when empty, and always before leaving, and also en route, to kill the rats that may be on board. A wise measure in international sanitation would be to require all vessels, whether trading at plague ports or not, to fumigate for rats no less than three or four times a year.

Food.—Rats are not strictly herbivorous, as might be inferred from their dentition; they are practically omnivorous. Their bill of fare includes grains and seeds of every kind; flour, meal, and all food products made from them; garden vegetables, mushrooms, bark of growing trees, bulbs, roots, stems, leaves, and flowers of herbaceous plants; eggs, chickens, ducklings, squabs, and young rabbits; milk, butter, and cheese; fresh meat and carrion; fish, frogs, mollusks, and crustaceans; they are also cannibals. This great variety of food explains the ease with which rats maintain themselves in almost any environment.

Habits.—The roof rat (*Mus alexandrinus*) and the black rat (*Mus rattus*) are more expert climbers than the brown rat, which is larger and clumsier. In buildings the brown rat keeps mainly to the cellar

and lower parts, where it commonly lives in burrows. From these retreats it makes nightly excursions in search of food. The roof rat and the black rat live in the walls or in the space between ceilings and roofs. Rats readily climb trees to obtain fruit. In the tropics the roof rat and the black rat habitually nest in trees. In the open rats seem to have defective vision; by daylight they move slowly and uncertainly; on the contrary, at the side of the room and in contact with the wall they run with great celerity. This fact suggests that the *vibrissæ* (whiskers) serve as feelers, and that the sense of touch in them is extremely delicate. The animals always prefer narrow places as highways —another circumstance which may be made use of in placing traps.

The ferocity of rats has been grossly exaggerated. The stories of their attacks upon human beings, sleeping infants especially, have but slight foundation. Ordinarily the probability of being bitten by rats is remote, and the bite is not usually poisonous. Miyake[1] has described a "rat-bite disease" called Sodoku in Japan.

Plague in Rats.—It is now known that rats are more or less responsible for cases of human plague, and in addition are the most frequent medium by which plague is carried from one locality to another. They also convey the plague infection to other rodents, such as ground squirrels.

The clinical manifestations of plague in rats are of little importance. It is generally said that a plague-infected rat staggers about with a drunken gait, loses fear of its natural enemies, and is readily captured. Rats experimentally infected show no marked manifestations of illness until shortly before death, when they become quiet, crouch in the corner of the cage, and try to hide. It is rather surprising that comparatively few plague rats are found dead in endemic centers. In the San Francisco campaign McCoy estimates that certainly not more than 20 per cent. of the infected rodents were found dead, the remainder being trapped. This is probably due to the fact that plague in rats is of several days' duration, and during this period there are good chances of catching the sick rodent in a trap, while the chance of finding the body after death is handicapped by obvious circumstances.

Rats suffer both with acute plague and chronic plague, the lesions of which differ.

The diagnosis of plague in rats may be made macroscopically. The Indian Plague Commission, which had the opportunity of examining an enormous number of plague rats in Bombay and elsewhere in India, state that "the results of tests carried out for the purpose of comparison make it manifest that the naked eye is markedly superior to the microscopic method as an aid in diagnosis, and as the result of our experi-

[1] *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1902; also Proescher, *Internat. Clinics*, IV, 25th Series, p. 77.

ence we are prepared to make a diagnosis of plague on the strength of the macroscopical appearance alone, even though the other results of cutaneous inoculation and culture are negative and the animals show signs of putrefaction." The experience of McCoy and others in the Federal Plague Laboratory in San Francisco leads to the same conclusion. It should be remembered, however, that occasionally plague occurs in rats without gross lesions. This has been observed by Dunbar and Kister and also by McCoy. In any critical case the bacteriological confirmation is essential.

Acute plague in rats is characterized by engorgement of the subcutaneous blood vessels and a diffuse pink color of the subcutaneous structures and muscles. The diagnosis may often be inferred at the first incision. The lymphatic glands of the neck, axilla, groin, or pelvis are enlarged and frequently surrounded by a hemorrhagic exudate and edema. The liver is granular with focal necroses, the spleen enlarged and friable, and pleural effusions are common.

Chronic plague in rats has been encountered in a considerable number of cases among *Mus rattus* in the Punjab villages of Kasel and Dhand. It has not been found in California. In the chronic disease the lesions consist of purulent or caseous foci, usually of the visceral type; that is, they occur as splenic nodules and abscesses, or mesenteric abscesses. Sometimes the abscesses are situated in the regions of the peripheral lymph glands. Plague bacilli are either absent or very scanty upon microscopic examination in these abscesses, but they may be recovered by cultural methods or more surely by inoculating the material into susceptible animals. There is no evidence to show that chronic rat plague has anything to do with the recurrence of acute plague among the rats.

Rats may be infected by the ingestion of infective material or the application of virulent plague bacilli to a mucous or cutaneous surface, or by subcutaneous injection of the microorganism. The infection may also be transferred from rat to rat through the agency of the flea. In nature the mode of transference probably takes place through all of these methods, but commonly through the flea.

Contrary to the general impression, the wild rat has a considerable resistance to plague infection. The Indian Plague Commission found that 59 per cent. were immune when inoculated by the subcutaneous method from the spleen of infected rats. A series of experiments conducted in the Federal laboratory in San Francisco also showed a high grade of immunity, especially among the large rats. About 15 per cent. of small rats and about 50 per cent. of large rats were found to be immune when inoculated with highly virulent material. The experiments demonstrated that this immunity is not acquired through a prior attack of the disease, but must be a natural immunity.

The natural subsidence of plague among rats in any community is a point about which much more evidence must be obtained before we can speak with any degree of authority. It may be due to a lack of susceptible material, possibly to a loss of virulence of the organism, but it seems more probable that it is due to a change in the number or relations of the ectoparasites of the rat.

Rat Leprosy.—Leprosy occurs spontaneously among rats and bears a close resemblance to the disease in man, but it seems that the rat leprosy is not communicable to man. For a further discussion of rat leprosy see page 293.

Trichinosis.—The three most important hosts for the *Trichinella spiralis* are man, swine, and rats. The infection is spread by one animal eating the flesh of another. It is, therefore, evident that if the disease occurred only in hogs and man it would soon die out. Rats, on account of their habits, may then be viewed as the great reservoir for the parasites and for the disease it causes. Hence, a well-directed public health campaign against trichinosis should consider the eradication of rats, especially around slaughter houses, butcher shops, hog pens, and similar places.

Trichinosis is very common among rats; they become infected by eating each other, by eating scraps of pork found on the offal pile of slaughter houses, butcher shops, or in swill. Swine become infected by eating rats and infected offal. Man becomes infected almost exclusively by eating pork or boar meat that has not been thoroughly cooked.

Other Parasites.—Rats and mice may harbor eleven species of internal parasites which also occur in man. Seven of these are of academic importance only. Those which concern us principally, in addition to the *Trichinella spiralis,* are the *Hymenolepis diminuta* and *Lamblia duodenalis.* Rats also harbor the *Cysticercus cellulosæ,* and are susceptible to experimental infections with *Trypanosoma gambiense,* the cause of sleeping sickness.

Rats have also been accused of dragging typhoid from the sewers to our food. The connection is close and the possibility apparent. A recent outbreak of typhoid fever in an asylum has, in fact, been traced to this source by Dr. Mills.[1]

Economic Importance.—The destruction of food, merchandise, and property by rats is so great that this alone would justify active measures of suppression, even though they were not responsible for plague, trichinosis, and other infections. Rats destroy grain while growing; invade stores, destroy flowers, laces, silks, carpets; eat fruits, vegetables, meat, etc., in the market; destroy by pollution ten times as much as they eat; cause conflagration by dragging matches into their holes; gnaw lead pipes and floors of houses; ruin artificial ponds and embankments by

[1] *Brit. Med. Jour.,* January 21, 1911.

burrowing; destroy eggs and young poultry; damage foundations, floors, doors, piers; in short, they have become the worst mammalian pest among us. It is estimated that in the United States alone the losses due to rat depredations vary from $35,000,000 to $50,000,000 annually.

Suppression.—The extermination of the rat is hopeless; they are very intelligent and cautious. Extermination seems a biological impossibility, for killing off large numbers gives the survivors an easier living. Millions of rats have been killed in India, Japan, San Francisco, and other places during the recent plague measures without making an appreciable impress upon the numbers remaining. They may be exterminated and kept out of a limited area, such as a ship, a granary, a stable, a warehouse, a market, or local compound. In the well-built residential sections of a city, with concrete walks, asphalt streets, stone cellars, and few stables, there are very few rats. In 10 years of residence in such a district in Washington I never saw or heard of one in the neighborhood.

The measures for the repression and destruction of rats will be considered under: (1) rat-proof buildings, (2) keeping food from rats, (3) natural enemies, (4) traps, (5) poisons, (6) domestic animals, (7) shooting, (8) fumigation, and (9) bacterial viruses.

Rat-proof Buildings.—This is a measure of first importance in the fight against rats. Rats can only gain entrance to a cement structure properly constructed through neglect or ignorance. They come in through drain pipes if left open; through doors, especially from alleys; and through basement windows. Once in, they intrench themselves in out-of-the-way places, nest behind rubbish, and are difficult to dislodge. The lower parts of the outer doors of public structures, such as markets and wharves, should be reinforced with metal to keep the rats from gnawing through. Basement windows should be screened and doors provided with springs to keep them closed.

A rat-proof dwelling must have concrete footings and the walls of a wooden house should have one foot of concrete between the sheathing and lathing. All water and drain pipes should be surrounded with cement. Rat holes may be closed with a mixture of cement, sand, and broken glass, or sharp bits of crockery and stone.

Aside from dwellings, the chief refuges for rats in cities are sewers, wharves, stables, provision houses, markets, out-buildings, and uninhabited structures. Modern sewers are highways and not nesting places for rats. They find a safe retreat from nearly all enemies under wooden sidewalks. In the country it is important to build corn cribs, barns, and granaries rat-proof with the liberal use of cement, iron sheeting, or galvanized iron netting.

Keeping Food from Rats.—Well-fed rats mature quickly, breed

often, and have large litters. A scarcity of food helps all other suppressive measures. Garbage and offal must be disposed of so that rats cannot get at such stuff. Well-covered garbage cans should be required and the garbage frequently removed and burned. To deposit it upon the ground anywhere only invites and nourishes rats and other vermin. Slaughter houses are centers of rat propagation. The offal is best disposed of by burning. Care should also be taken as to the disposal of remnants of lunches in office buildings and the disposal of organic waste generally. Produce in provision stores may be protected with wire cages.

Natural Enemies.—The natural enemies of the rat are the larger hawks, owls, skunks, foxes, coyotes, weasels, minks, dogs, cats, and ferrets. The persistent killing off of the carnivorous birds and mammals that prey upon rats has been an important factor in the increase of these rodents in the United States. Rats actually destroy more eggs, chickens, and game than all the wild animals combined.

Traps.—There are many kinds of traps, such as the guillotine, spring trap, the cage trap, the barrel and pit trap. One of the best is the old-fashioned wire cage trap. The rats get in but cannot get out. In placing the trap it is advisable to leave a rat in as a decoy. The trap should be placed along runways, or the entrance to the trap may be arranged so that the rats first have to go through a pipe, as they like to explore dark passages. It requires ingenuity to successfully trap rats. They are very wary and avoid man-smell. To guard against this the traps may be burned and then smeared with the bait, always handling them with tongs or properly prepared gloves. Cheese, bacon, grain, and bread are the best baits.

Poisons.—Poisons are objectionable in dwellings, owing to the odor of the dead rats. They are of service in granaries, stables, wharves, and similar places. Most rat poisons are dangerous to children as well as to chickens and other domestic animals, and, therefore, the greatest care must be exercised in their use. It requires experience in laying out poisons; the old rats are very smart and will refuse the bait unless artfully concealed and judiciously placed.

The principal poisons used for rats are barium carbonate, strychnin, arsenic, and phosphorus. In several states the law requires that notice of intention to lay poison must be given to persons living in the neighborhood. Poisons for rats should never be placed in open or unsheltered places. For poisoning rats in buildings and yards occupied by poultry the following procedure is recommended: Two wooden boxes should be used, one considerably larger than the other, and each having two or more holes in the sides large enough to admit rats. The poisoned bait should be placed in the bottom and near the middle of the smaller box, and the larger box should then be inverted over the other. Rats thus have free access to the bait, but fowls are excluded.

The cheapest and most effective poison is barium carbonate. This may be made into a dough with four parts of meal or flour to one part of barium carbonate. A good plan is to spread the barium carbonate upon fish, on toasted bread (moistened), or upon ordinary bread and butter.

Strychnin is effective and may be used by inserting the dry crystals in a piece of meat, cheese, or sausage, which is placed in the runways.

Arsenic is popular; the powdered white arsenic (arsenious acid) may be used as described for strychnin or barium; or a stiff dough may be made by mixing twelve parts by weight of corn meal and one part of arsenic with whites of egg. An old English formula is one pound of oatmeal, one pound of brown sugar, and a spoonful of arsenic.

Phosphorus is an effective and attractive bait. The yellow phosphorus in the proportion of one to four per cent. may be mixed with glucose or other suitable material. The use of phosphorus is very dangerous on account of fire. Rats poisoned with phosphorus may die on the premises and decompose, contrary to the statements sometimes made in the advertisements.

The following formula is recommended as a poisonous bait for rats, mice, squirrels, etc.:

Strychnin	1 oz.
Cyanid of potassium	2 oz.
Eggs	1 doz.
Honey	1 pint
Wheat or barley	30 lbs.

Stir eggs well, then mix in honey and again stir. Then put in dry powdered strychnin and cyanid and stir until well mixed. Put wheat in large box or can and pour in the mixture of poison and stir until it is well distributed over the wheat. Stir two or three times during twenty-four hours, then spread out and dry. Before putting it out for squirrels add oil of rhodium, 1 drachm.

Domestic Animals.—A well-trained dog may be relied upon to keep the farm premises reasonably free of rats. Small Irish, Scotch, and fox terriers make the best ratters; the ordinary cur and the larger breeds of dogs seldom develop the necessary qualities for ratters.

However valuable cats may be as mousers, few of them learn to catch rats. The ordinary house cat is too well fed and too lazy to undertake the capture of an animal as formidable as the brown rat. Koch has advised the breeding and distribution of cats capable and willing to attack rats.

Shooting.—Many rats may be shot as they come out to forage about sundown. This method is particularly effective in a large building

which is suddenly overrun with the rodents. The shooting of a number of them upon two or three successive nights discourages the remainder, who leave for some other happier hunting ground.

Fumigation.—Rats may be killed with certainty in any inclosed structure by the use of sulphur dioxid, carbon bisulphid, hydrocyanic acid gas, or carbon monoxid. The methods of evolving these substances have been described in Section XII. Sulphur dioxid is particularly useful to destroy rats on board ships, in cellars, stables, sewers, and places where they abound and which are not injured by the corrosive action of the sulphur fumes. Enormous numbers of rats are frequently killed when ships are fumigated with sulphur dioxid. I have seen buckets full thrown overboard from comparatively small vessels. Hobdy counted 310 on a lumber-carrying schooner of only 260 tons burden. The S.S. *Minnehaha,* a new vessel only nine months in commission fumigated in London in May, 1901, yielded a bag of 1,700 rats.

For the destruction of rats upon vessels the sulphur dioxid may be produced by the pot method, if the hold is empty, or may be generated in a Kinyoun-Francis or a Clayton furnace, or may be liberated from its compressed liquefied state. No less than three pounds of sulphur should be burned for each 1,000 cubic feet of space, and the exposure should not be less than 5 hours (see page 997).

Carbon Monoxid.—Carbon monoxid is an exceedingly poisonous gas. From the fact that it has no odor it is even more hazardous in practice than hydrocyanic acid. Carbon monoxid is fatal to all forms of mammalian life, but has no germicidal properties whatever. It has been used in Hamburg[1] and other ports for the destruction of rats on ships.

Carbon monoxid is a colorless, odorless gas, lighter than air. It forms a stable compound with the hemoglobin of the blood—carbon monoxid-hemoglobin. For the toxic action of this gas and its other properties see page 637. The particular advantages of carbon monoxid for the destruction of rats on board ship are that it may be generated cheaply, is quickly effective, and does no injury to cargo or vessel. The disadvantages are that it is poisonous and inflammable. The addition of a little sulphur dioxid to the gas makes its presence known and tends to prevent accidents. After exposure the hold must be thoroughly ventilated, and it is customary to lower a mouse in a cage for 10 minutes to be sure that it is safe for a man to enter. Divers' helmets should also be kept in readiness so that the hold may be entered in case of need.

A gas generator has been made by Pintch which furnishes a mixture consisting of CO, 5 per cent., CO_2, 18 per cent., N, 77 per cent. These gases are generated by the incomplete combustion of coke. The

[1] Nocht and Giemsa: *Arbeiten a. d. kaiserlichen Gesundheitsampte,* Bd. 20, Ersten Heft, 1904, p. 91.

mixture of gases is pumped into the hold of the vessel or other compartments where it is desired. The hold should be kept tightly closed from 7 to 8 hours.

The Bacterial Rat Viruses.—Rats are notoriously resistant to bacterial infection.[1] Even plague usually fails markedly to diminish their prevalence. An epizootic of bacterial nature, therefore, cannot be classed with the natural enemies of the rat. We are not surprised, then, to learn that the bacterial rat viruses have signally failed to accomplish their mission.

These bacterial viruses belong to the colon-typhoid group of organisms. They are either identical with or closely related to the original bacillus of mouse typhoid (*B. typhi murium*) discovered by Loeffler, or the paratyphoid bacillus, type B, which is frequently the cause of meat poisoning, or the *Bacillus enteritidis* of Gaertner, which has been associated with gastrointestinal disorders.

The claim that these rat viruses are harmless to man needs revision, in view of the instances of sickness and death reported by various observers. The pathogenicity for man depends upon the virulence of the culture, the amount ingested, the nature of the medium in which it grows, and many other factors.

Danysz virus (*B. typhi murium*) is pathogenic for rats under laboratory conditions, but has feeble powers of propagating itself from rat to rat. It rapidly loses its virulence, especially when exposed to light and air. The result depends largely upon the amount ingested. The other viruses have proven even less satisfactory.

Under natural conditions these rat viruses may be likened to a chemical poison, with the great disadvantage that they rapidly lose their virulence and are comparatively expensive. They also have the further disadvantage that chemical poisons do not possess of rendering animals immune by the ingestion of amounts that are insufficient to kill or by the ingestion of cultures that have lost their virulence.

Squirrels.—In August, 1903, a blacksmith died of plague probably contracted from a squirrel in Contra Costa County, California. In 1904 Currie demonstrated the susceptibility of the ground squirrel to bubonic plague. In 1908 McCoy and Wherry discovered natural plague in ground squirrels. It was then learned that thousands of squirrels had died of some disease during 1904, 1905, and 1906. This epizoötic was doubtless plague. It is now realized that plague has become endemic in California, in the squirrel. It is also believed that the disease has been kept alive in the endemic foci of Tibet in another rodent, the marmot (*Arctomys bobac*). The eradication of plague

[1] "The Inefficiency of Bacterial Viruses in the Extermination of Rats," M. J. Rosenau. "The Rat and Its Relation to the Public Health," *Bulletin* of the P. H. & M. H. S., 1910.

must, therefore, consider these and perhaps other susceptible wild animals.

California is overrun with three species of ground squirrels. The commonest is the *Citellus beecheyi*. They live in colonies in burrows or warrens. The burrowing owl is a frequent companion occupying the same burrow, and they probably spread the infection by carrying fleas. Squirrels become infected through fleas from each other and from rats. The squirrel flea (*Ceratophyllus acutus*) attacks man just as the rat flea does. The infection may also be conveyed to man through squirrel bites, as in the case of the child in Los Angeles studied by Stimson. Squirrels make good food for man, but since the danger has been realized the shooting or trapping of them for food purposes is now forbidden in California.

Plague in the squirrel may be recognized[1] by the gross anatomical lesions in the lymphatic glands, the liver, and lungs. The pneumonic form of the disease is common in the squirrel. Many cases are subacute or chronic. Smear preparations from squirrels dead of plague are frequently negative for plague-like bacilli. The diagnosis may, therefore, be made more surely by animal experimentation. Subcutaneous inoculation is surer than the cutaneous method, as the latter often fails on account of the comparatively few plague bacilli present in squirrel lesions.

Squirrels may be destroyed by various means. One of the most successful is to saturate cotton waste the size of an orange with carbon bisulphid and place it in the warren; then close the opening with wet clay. Poisoned bait, such as strychnin, phosphorus, or cyanid of potassium, is effective. Traps are not very successful, as the squirrel is wary. Natural enemies, such as the coyote, wolf, badger, skunk, mountain lion, the cobra snake, and red-tailed hawk should be encouraged.[2]

PLAGUE

In considering the prevention of plague it is necessary to recognize that the different types of the disease are spread in different ways. At least three clinical types are now recognized: (1) bubonic, (2) pneumonic, and (3) septicemic.[3] In the bubonic and septicemic types of the disease the plague bacillus is locked up in the glands, blood, and other tissues and organs of the body, and are not eliminated

[1] McCoy: *Jour. of Infect. Dis.*, Nov. 26, 1909, Vol. V, No. 5.

[2] In this chapter material has been freely drawn from "The Rat and Its Relation to the Public Health," Public Health and Marine Hospital Service, 1910, particularly articles by Lantz, McCoy, Brinckerhoff, Banks, Stiles, Rucker, Creel, Holdy, Kerr, and Rosenau. This book may be obtained by addressing the Surgeon-General or the Superintendent of Public Documents, Washington, D. C.

[3] Occasionally other varieties occur in which the chief manifestations are in the skin and subcutaneous tissues, or in the intestines, causing diarrhea. In the latter case the infection is excreted in the feces.

in the usual excretions. These forms of the disease are, therefore, not "contagious," but are spread mainly through the agency of the flea. On the other hand, in the pneumonic type of the disease plague bacilli are contained in enormous numbers in the sputum. The disease is frequently transmitted directly by close association with a patient having plague pneumonia. The pneumonic type of the disease does not necessarily follow when the infection is taken into the system through the respiratory channel; on the other hand, it may result from infection through a flea bite.

The *Bacillus pestis* (Yersin, 1894) has more than fulfilled Koch's laws. Several accidents in which pure cultures have been inoculated into man, producing all the symptoms and lesions of the disease, have added to the proof that this organism is the cause of plague (Vienna, 1898, Ann Arbor, 1902, and also in laboratories in Russia, Berlin, and Japan). The plague bacillus is comparatively easy to isolate and grows readily on artificial culture media, and has characteristics that readily distinguish it from all other species. It is a short rod with rounded ends, not motile, decolorized by Gram's method, and grows better at 30° C. than at blood temperature.

Recognition of the plague bacillus rests upon the following characteristics: (1) Curious involution forms upon salt agar within 24 hours; (2) stalactite growth in liquid media; (3) characteristic lesions produced by experimental plague in guinea pigs, rabbits, rats, etc. Kolle's method consists in rubbing the material containing the plague bacillus upon a shaved area of the skin of a guinea pig. The plague bacilli penetrate the skin, leaving other pathogenic organisms behind. The skin of the guinea pig thus acts as a differential filter; (4) the final test of the identity of the plague bacillus is the fact that its pathogenicity may be neutralized by the use of antiplague serum.

The *Bacillus pestis* does not live a saprophytic existence in nature. It is readily killed by drying, sunlight, heat, and the usual germicides. The organism does not live long in the soil or upon the floors of houses, as was once commonly supposed. There is, therefore, comparatively little danger from these sources.

Immunity.—One attack of plague usually protects for life. Occasionally second attacks are noted in the same person. In such cases the second attack is usually mild. This is an old observation and led to the employment of persons with a plague history or a plague scar in hospitals and laboratories.

Artificial immunity of either an active or passive nature may be acquired by various procedures. The passive immunity produced by the injection of antiplague serum lasts only about three to four weeks. The active immunity produced by vaccination of cultures may be depended upon for about six months.

Haffkine's prophylactic consists of a killed culture of the plague bacillus, which is injected subcutaneously. Haffkine used a bouillon culture, six weeks old, grown at 25-30° C. and killed at 65° C., for one hour. One-half of one per cent. of phenol is then added. From 2 to 3.5 c. c. (this was later increased to 20 c. c.) of this vaccine are injected subcutaneously. Ten days later a second injection of a still larger amount is given.

In twelve districts in India 224,228 persons were inoculated with Haffkine's prophylactic. Of these 3,399 took the disease. Of 639,600 not inoculated in the same districts 49,430 were attacked. C. J. Martin concludes that the chances of subsequent infection are reduced four-fifths, and the chances of recovery are 2.5 times as great as in the cases of the non-vaccinated.

The German Plague Commission prepared their prophylactic vaccine from a fresh virulent agar culture, suspending the bacilli in salt solution or bouillon. The organisms are killed at 65° C. for one to two hours, and 0.5 per cent. phenol added. The amount injected represents one agar culture.

Lustig and Galliotti extract the immunizing substance from the bacterial cell (endotoxin) with weak potassium hydroxid. This nucleoprotein is collected and dried, and thus permits of exact dosage. The amount injected is two to three milligrams of the dry extract dissolved in water.

Terni and Bandi recommend the peritoneal exudate of plague-infected guinea pigs, sterilized fractionally at 50° C., and the addition of 0.5 per cent. of phenol, 0.25 per cent. sodium carbonate, and 0.75 per cent. sodium chlorid.

Shiga prefers a combined active and passive immunity produced with killed cultures and antipest serum, because this mixed immunizing process has the advantage of producing milder reactions.

Kolle and Strong started out from the principle that a much higher degree of immunity is produced by living microorganisms than dead ones, and recommend the use of live attenuated cultures. Strong has a strain, an entire agar culture of which may be injected into man without harm. In Manila 42 persons were given a preventive inoculation with this culture.

The reactions which follow vaccination with a plague culture, whether alive or dead, are sometimes marked. The symptoms consist of a rise in temperature to 39° C., malaise, depression, and headache, and swelling and pain at the site of the inoculation. The symptoms usually pass away in 24 to 48 hours.

The production of an active acquired immunity has a distinct practical usefulness in the prevention of the disease, although it cannot take the place of rat and flea eradication. It has been used on a large

scale by Haffkine in India, and to a lesser extent by others in many parts of the world during the recent plague pandemic. Those who get plague after Haffkinization usually have a mild form of the disease, which, in the experience in India, rarely results in death. The active immunization of the community in the face of an epidemic is a valuable addition to our preventive measures against plague. It is of first importance in protecting small communities, on shipboard, in camps and barracks, at quarantine stations, in plague laboratories, among rat brigades, as well as for physicians, nurses, and others who are exposed.

Yersin's serum is obtained from a horse that has received repeated injections of plague cultures; at first killed plague cultures, afterward living bacilli, are used. At most this antitoxic serum is weak, and, while it has a certain amount of protective properties, it has slight curative power. Very large quantities must be administered early in the disease to obtain any effect at all. The protection lasts only a few weeks, three to four at most, and is, therefore, of limited practical use.

Endemic Foci.—There are four historic endemic foci in which plague has slumbered for ages. One is on the eastern slope of the Himalayas, in the valley of the Yünnan. The great epidemic in Hongkong in 1894 came from this center. A second endemic focus near, and perhaps connected with the first is on the western slope of the Himalayas. From here the infection was carried to Bombay in 1896, where it still prevails. A third plague focus exists from about the center of Arabia to near Mesopotamia. From here the infection was dragged to Samarkand, the Black Sea, and Persia. The fourth endemic area was discovered by Koch in 1898 in the interior of Africa, near the source of the White Nile in Uganda. We must now add to this a fifth endemic focus, for plague has obtained a foothold in California in the ground squirrels, which will take years of well-directed energy to control. The disease has caused dreadful havoc in India since 1892. In 1907 over one million persons died of plague in that country.

Plague is kept alive in the endemic foci in the rat, the ground squirrel, the marmot, the brush rat, and other rodents. The campaign of eradicating plague in the ground squirrel in California has been directed to killing as large a number of these animals as possible. For this purpose carbon disulphid, sulphur dioxid, hydrocyanic acid gas, bait poisoned with strychnin, and cyanid of potassium are used. Natural enemies, such as the coyote, wolf, fox, badger, snake, mountain lion, skunk, and red-tailed hawk, are encouraged. Trapping has not proven successful, as the squirrel is a very wary animal. Many squirrels may be shot, but those killed should not be used for food.

Management of a Plague Epidemic.—The handling of a plague epidemic is conducted along two definite lines of activity. One is to

find and care for the human cases, the other consists in a warfare against rats. The organization and general management of a plague campaign do not differ radically from similar work in other epidemics (see page 319). Cases of the disease must be sought for and early diagnosis confirmed; all deaths from no matter what cause must be investigated, and the body examined by an expert before burial is permitted. A bacteriological laboratory is a *sine qua non*. Cases of the disease should be isolated and the usual disinfection of excreta and surroundings exercised. Particular care must be taken that the isolation wards are vermin-free. The place from which the case is removed should then be given a preliminary disinfection with sulphur dioxid or other substance that may be depended upon to kill rats and fleas, and a search made in the neighborhood for secondary cases both in man and rodents.

The campaign against the rat is expensive and difficult, but must be vigorously prosecuted to insure success. The rat warfare may be briefly summarized as a simultaneous attack upon the habitation and food supply of the rat; the destruction of rat burrows and nesting places; the separation of the rat from his food supply by concreting and screening such places as stables, warehouses, markets, restaurants, etc.; the prevention of the entry of the rat into human habitations by the use of concrete, wire netting, or other barriers; and the use of poisons, traps, etc. For further consideration concerning rats and their eradication see page 242. All the rats that are caught in traps or found dead are brought to the bacteriological laboratory, where they are examined and careful records kept concerning the species, the location, the place where the rat was caught, the character of the infection, etc. As it is a hopeless task to exterminate rats from a large city, Heiser has proposed a practical plan which proved effective in Manila. A list of the places in which the plague-infected rats were found was made. Each was regarded as a center of infection. Radiating lines, usually five in number, were prolonged from this center, evenly placed like the spokes of a wheel. Rats were caught along these lines and examined. Plague rats were seldom found more than a few blocks away. The furthermost points at which the infected rats were found were then connected with a line, as is roughly shown in the diagram, Figs. 38 and 39. The place inclosed by the dotted line was regarded as a section of infection. The entire rat-catching force was then concentrated along the border of the infected section, that is, along the dotted line. They then commenced to move toward the center, catching the rats as they closed in. Behind them ratproofing was carried out. One section after another was treated in this way, until they had all been wiped out. Once weekly thereafter rats were caught in the previously infected sections and at other places, especially those which had been

infected in years gone by. Since the above system was adopted plague has disappeared in the city of Manila, and at a cost of only a small fraction of that of a general rat extermination campaign.

Quarantine.—Plague infection is frequently carried over seas in vessels. When this happens it is more apt to be due to the disease in the rat than man. Maritime quarantine, therefore, finds its greatest justification in keeping out plague. To be successful, measures must be directed almost entirely against the rat, although a keen eye must

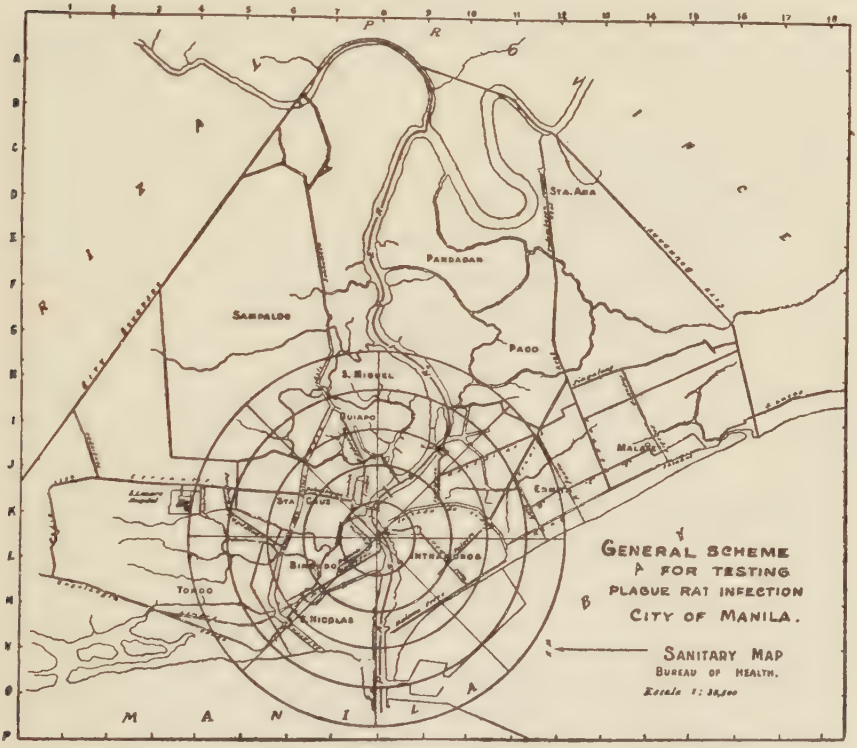

FIG. 42.

be kept on the lookout for mild cases known as *Pestis minor,* or walking plague. Rats may be kept down on board a vessel by the frequent use of sulphur dioxid. All vessels trading with a plague-infected port should have each cargo compartment fumigated with this gas, at least when it is empty, at the port of departure. The vessel must be again fumigated with sulphur dioxid on arrival. Both at the infected port and at the port of arrival care must be taken to prevent the ingress and egress of rats. The period of detention of the personnel for a plague ship is seven days. For further details concerning quarantine see page 321.

Prevention—Summary.—The principles and many of the details for the prevention of plague have been stated in the foregoing pages, and need not be repeated.

Personal prophylaxis consists in avoiding the infected regions and guarding against flea bites. Physicians and nurses should remember that the pneumonic form of the disease is highly "contagious" in the ordinary sense of the term. Attendants and persons who come in contact with such cases may protect themselves with Haffkine's prophylactic or Yersin's serum. Individual measures to guard against droplet

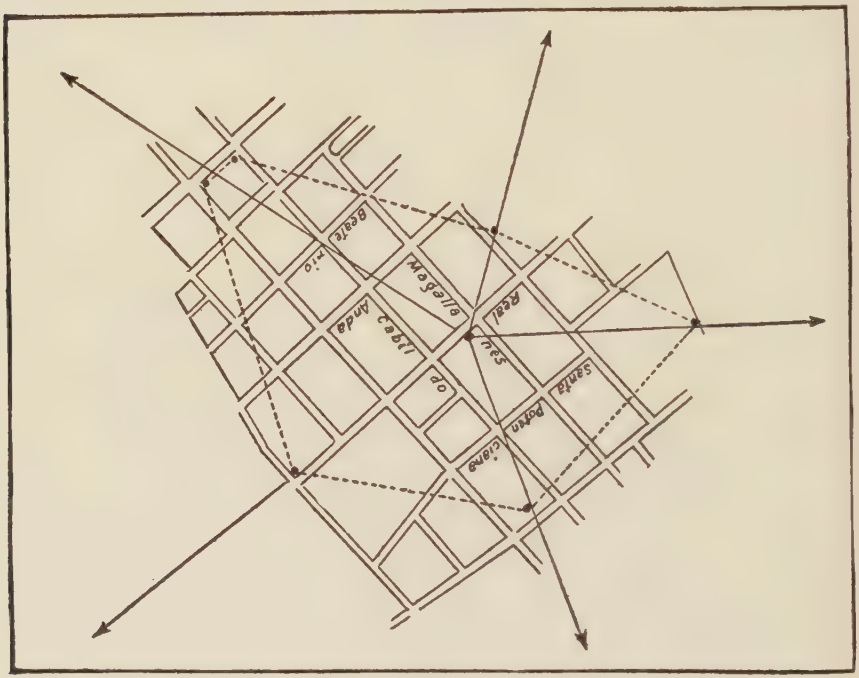

Fig. 43.—Isolated Plague-infested Center, Manila, P. I.

infection, such as the wearing of masks or veils of cheesecloth, may be resorted to. The bubonic and septicemic forms of the disease are not, as a rule, directly communicable, and, therefore, the preventive measures recommended for typhoid fever are effective.

The ordinary germicidal solutions, such as bichlorid of mercury, 1-1,000, carbolic acid, 2½ per cent., formalin, 10 per cent., are effective against the *Bacillus pestis*. Of the gaseous disinfectants sulphur dioxid is preferred, because it not only kills the frail plague bacillus, but also destroys rats, fleas, etc.

Cases of plague should be isolated in a well-screened room otherwise free of insects. Fabrics and other objects which become contam-

inated with the discharges should be thoroughly disinfected by proper methods.

It is important to have prompt reports of all cases of suspected plague, and the diagnosis must be confirmed by bacteriological methods. In all plague centers there should be a special hospital and also a laboratory where diagnostic work may be carried on; this is an essential part of the equipment for a successful campaign. A traveling laboratory organized like a flying squadron for quick service should be provided to furnish this service wherever it may be demanded.

The prevention of plague, after all, is reduced to warfare against rats and fleas. This has been fully discussed. All seaport towns having communication with plague countries would do well to examine for plague rats caught from time to time about the wharves. This, in fact, should be one of the routine duties of the port sanitary authorities. Plague may slumber in the rats for years before human cases occur. Other preventive measures are obvious from the nature of the infection and its mode of transmission, or have already been stated in the preceding pages.

TICKS

Ticks belong to the family Ixodidæ, and the diseases which they transmit are known as ixodiasis. Quite a number of different species are known to attack man.

Ticks, or wood lice, are not true insecta, but belong to the acarines which include the mites, and are closely allied to spiders and itchmites (scabies). Ticks have an unsegmented body with eight legs in the adult stage and six legs in the larval stage. In some of their habits they resemble bedbugs. So far as is known, they take no vegetable food, but live on blood. Ticks are ectoparasites of man and many animals. They frequently hang tenaciously to the skin, in which they partly bury themselves. If covered with oil or vaselin, thus closing their breathing pores situated behind the fourth pair of legs, they may be induced to release their hold. If pulled off roughly the head (capitulum) is likely to break off and remain in the skin. Sulphur in some form is useful to destroy ticks in the adult stage. Sulphur ointment is particularly obnoxious to this group of parasites. Arsenic and crude oil also act as poisons to the tick, and may be used by local application.

The life cycle of the tick consists of four distinct stages, viz.: egg (embryo), larva, nymph, and adult. The eggs are invariably deposited on the ground in large masses. The larvæ which emerge from the eggs are minute six-legged creatures. The larvæ attach themselves to a suitable host, upon which they feed, then usually drop to the ground

and molt, becoming nymphs. The nymphs have eight legs. The nymph waits until it can attach itself to a host, engorges blood, usually drops, molts its skin, and becomes adult. The life history of the tick differs from the mosquito in that the larval and pupal stages are not aquatic.

It was first shown by Smith and Kilborne that in the case of Texas fever the microorganism within the adult tick passes into the egg and is, therefore, transmitted "hereditarily" to the next generation. The infection of Rocky Mountain spotted fever, of canine piroplasmosis, and probably also that of African tick fever, is also transmitted by the female to the next generation. Tick-borne diseases are not always transmitted in nature in this way. The virus may be transferred directly by the larva, the nymph, or the adult. Thus some ticks leave their host repeatedly, and the parasites they draw from one animal may be injected into another animal either during the same or at a subsequent stage in the development of the tick.

Ticks upon domestic stock may be controlled by dipping, spraying, or by hand methods. The arsenical dip has practically displaced all others for the destruction of ticks in the various parts of the world. Crude oils have been used to a considerable extent in some cases. They are more expensive than the arsenical dip, and dangerous to cattle under some conditions. Serious losses have followed the use of heavy oils in dry regions, or where it has been necessary to drive the cattle any considerable distance after dipping.

The formula for the arsenical dip is as follows:

Sodium carbonate (sal. soda)	24 lbs.
Arsenic trioxid (white arsenic)	8 lbs.
Pine tar	2 gals.
Water	sufficient to make 500 gals.

Sometimes dipping is not practical. Instead of driving cattle considerable distances to dipping vats it will be found sufficient to treat them thoroughly by hand methods. The procedure consists simply in applying the arsenical mixture liberally by means of rags, mops, or brushes, or by means of spray pumps. Crude oil may be used by hand instead of the arsenical solution. For most tick-borne diseases cattle must be dipped or treated weekly.

The following diseases transmitted by ticks will be given brief consideration: Texas fever (*Margaropus annulatus*), South African tick fever (*Ornithodoros savignyi*), Rocky Mountain spotted fever (*Dermacentor venustus*), and relapsing fever (*Ornithodoros moubata*). Although it is probable that the latter disease is also transmitted by the *Argas persicus,* and perhaps other biting insects.

TEXAS FEVER

Texas fever or splenetic fever is also known as bovine malaria, tick fever, and hemoglobinuria. The disease does not affect man. It is confined to cattle, and is of very great economic importance. Texas fever is an infection which should be understood by all sanitarians, on account of its scientific and historic importance. The cause of this infection and its mode of transmission were ascertained in 1893 by Smith and Kilborne. The discovery that the tick is the intermediate host of Texas fever opened an entirely new principle in the sanitary sciences.

Texas fever is caused by a protozoon parasite. This parasite was first named *Pyrosoma bigeminum* on account of the twin-like, pear-shaped forms commonly seen in the red corpuscles. This genus was changed by Patton in 1895 to *Piroplasma*. These terms having been preoccupied, the present name of the parasite is *Babesia bigemina*.[1]

FIG. 44.—THE TEXAS FEVER TICK (*Margaropus annalatus*).

The contagium is carried by the cattle tick, *Boophilus bovis,* now *M. annulatus*. The tick lives upon the skin and feeds upon the infected blood, becomes sexually mature at the last molt; the female drops to the ground and lays about 2,000 eggs; the newly hatched larvæ attach themselves to the skin of a fresh host, which they infect. This explains the long extrinsic period of incubation in this disease, 40-60 days; 30 days of which are required for the development of the larvæ and the remainder for the development of the parasite within the host.

ROCKY MOUNTAIN SPOTTED FEVER

This disease, also called tick fever and spotted fever, is an interesting infection which occurs chiefly in the Bitter Root Valley of Montana, centering around Missoula. Cases also occur in the neighboring states

[1] These various names are given for the reason that they are all found in the literature.

of Idaho and Wyoming. The symptoms closely resemble those of typhus fever, including a petechial eruption. Anderson and Goldberger have shown that typhus fever of Mexico, called tabardillo, is not trans-

FIG. 45.—ROCKY MOUNTAIN SPOTTED FEVER TICK. (*Dermacentor venustus*).

1, Adult female, unengorged, dorsal view; 2, Adult male, dorsal view; 3, Adult female, unengorged, ventral view; 4, Adult male, ventral view; 5, Adult female in act of depositing eggs.

missible to guinea pigs, while Ricketts and also King independently demonstrated that some of the infected blood of a case of Rocky Mountain fever injected into a guinea pig will reproduce the chief features of this disease. The two diseases are, therefore, distinct.

Wilson and Chowning first suggested that the tick acts as the carrier of Rocky Mountain spotted fever. This was proven by Ricketts in 1906, who showed that the particular tick is *Dermacentor occidentalis* (now *venustus*). The infection may be transmitted by the larva, the nymph, and both the adult male and female ticks. The infection is also transmitted hereditarily through the ticks to their larvæ. The disease has been transmitted through the tick from man to monkey and the guinea pig, and also from monkey to monkey and from guinea pig to guinea pig. A few infected ticks have actually been found by Ricketts in nature.

Mayer [1] has proved by experiment that different species of ticks collected from various regions [*Dermacentor marginatus* (Utah), *Amblyomma americanus linnaus* (Missouri), and *Dermacentor variabilis* (Mass.)] are able to transmit the virus of Rocky Mountain spotted fever. The inference is that the disease may find favorable conditions for its existence in localities other than those to which it is now limited.

One attack of the disease establishes a rather high degree of immunity to subsequent attacks. The blood serum of recovered cases contains protective properties of a rather high degree for guinea pigs. The virus is not filterable through a Berkefeld filter. The nature of the virus is not known.

The prevention of Rocky Mountain spotted fever is directed entirely against the tick. Ticks are to be avoided in the infected region. If it is necessary to work in the fields and woods and about animals where these ticks abound, the bites should at once be cauterized with strong carbolic acid. A vigorous campaign should be carried on by the health authorities to destroy all the ticks in and about each case of the disease.

The ultimate control of Rocky Mountain spotted fever depends upon the suppression of the *Dermacentor venustus*. This, perhaps, is not so hopeless a task as may at first seem likely.[2] Henshaw and Birdseye [3] found twenty species of five hundred mammals examined in and around Bitter Root Valley to carry ticks either in the immature or adult stage. The mammalian hosts of fever ticks fall naturally into two groups: those that harbor chiefly adult ticks and those that harbor the younger stages. In the former class belong mountain goats, bears, coyotes, badgers, woodchucks, and possibly elk, deer, mountain sheep, rabbits, and domestic stock, such as horses, cattle, and sheep. Those of the second class harboring the nymphs and larvæ are mainly rodents and comprise ground squirrels, woodchucks, chipmunks, pine squirrels, mice, and

[1] *Jour. Infect. Dis.*, April 12, 1911.

[2] Fortunately the *Dermacentor venustus* is the only tick in the endemic region which attacks man.

[3] U. S. Dept. of Agr., Bureau of Biol. Survey, *Cir. 82*.

wood rats. These smaller animals are too agile to permit the adult ticks to remain upon them.

Unquestionably the great bulk of fever ticks (*Dermacentor venustus*) which become engorged in the Bitter Root Valley do so on domestic stock—horses, cattle, sheep, and sometimes dogs. They obtain the ticks from the pastures and other uncultivated land infested by wild animals. It is obvious, therefore, that, if the domestic animals in the valley are rendered tick-free by dipping, spraying, or by some other equally effective method, the chances of the infection of human beings will be vastly lessened. Dipping carried on upon an extensive scale throughout the endemic area by McClintic has given surprisingly successful results, there having been fewer cases of the disease this season in the Bitter Root Valley than for any year of record.

Supplementary measures for the control of Rocky Mountain spotted fever consist in the reduction of the number of rodents and the clearing of the brush land along the edges of the valley.[1]

McClintic infected Rhesus monkeys and guinea pigs with spotted fever and treated them with the following drugs: salvarsan, sodium cacodylate, and urotropin. The results obtained, however, do not indicate that any of these drugs possess any value whatever either as a prophylactic or in the treatment of spotted fever, but, on the contrary, their administration seems on the whole rather to intensify the severity of the disease in the animals compared with the course of the disease in the controls.[2]

RELAPSING FEVER

Relapsing fever, also called famine fever and seven-day fever, is found upon all the five continents of the globe. Epidemics of this disease have been reported, especially from Ireland and Russia. The infection prevails in India, where Vandyke Carter of Bombay made his classic investigations. Relapsing fever was epidemic in New York and Philadelphia in 1869. It has not reappeared. The disease has receded from civilization where cleanliness is observed.

Obermeier in 1868 discovered the spirillum in the blood—*Spirillum obermeieri*. Carter and Koch in 1878 showed that the infection may be transferred to apes by the inoculation of the blood of a patient. Münch and Moczutkowski transferred the disease by the inoculation of relapsing fever blood to healthy individuals. Koch succeeded in demonstrating that the spirochetes of African relapsing fever multiplied in the tick (*Ornithodoros moubata*), and that the bite of this tick may convey the disease to healthy men. The African relapsing fever

[1] Hunter, W. D., and Bishopp, F. C.: "The Rocky Mountain Spotted Fever Tick," *Bureau of Entomology Bull. No. 105*, U. S. Dept. of Agr.

[2] *U. S. Pub. Health Reports*, Vol. XXVII, No. 20, May 17, 1912.

which Koch studied in East Africa shows some slight differences from the European disease.

Although Koch and also Dutton and Todd demonstrated that the African relapsing fever may be transmitted through the bite of a tick, it is very probable that in Europe and other countries where relapsing fever occurs the disease may also be transmitted by the *Argas persicus*. In fact, other insects, as bedbugs, fleas, biting flies, and lice, may convey the infection.

Leishman[1] has demonstrated that the *Spirochæta duttoni* may be transmitted hereditarily in the tick. He has obtained positive results in the second generation, the bites of which were infective for mice and monkeys. Attempts to carry the infection to the third generation in the tick have so far failed. Leishman considers the hereditary transmission of the infection as biological evidence that the spirochetes belong to the protozoa rather than the bacteria.

Schuberg and Manteufe[2] found that a temperature of 22° C. is not favorable for the spirochete in the *Ornithodoros moubata*. This was shown by experiments upon rats in which the infection through the bite of the tick disappeared more quickly at 22° C. than at higher temperatures.

One attack protects against subsequent attacks. Second attacks among negroes in Africa in after years are very light. The only susceptible animals are man, the apes, mice, and rats.

The prevention of relapsing fever is based upon personal and domestic cleanliness and the avoidance of tick and other bug bites. Personal prophylaxis depends upon keeping aloof from vermin-infested places, especially where the disease prevails. Manson suggests that a mosquito net, a bed well off the ground, and a night light are indispensable in Africa, where the nocturnal habits of the *Ornithodoros moubata* render the hours of sleep especially dangerous.

SOUTH AFRICAN TICK FEVER

This is a febrile disease common in parts of Africa. The incubation is from five to ten days and the attack lasts from two days to a week or more, with abdominal pains, chills, vomiting, diarrhea. Relapses do not occur as in relapsing fever. The disease is caused by a spirillum very similar to the *Spirillum obermeieri*, but shown by Novy and others to have slight differences. The spirillum was demonstrated in 1905 by Dutton, who also showed that the infection can be transferred to monkeys by the bites of young ticks at their first feeding after hatching from infected parents. Here again is an instance of the hereditary

[1] *Lancet*, Jan. 1, 1910, Vol. 1, p. 11.
[2] *Zeitschr. f. Immunitätsforschung*, Orig. Bd. 4, 1910, p. 512.

transmission of the parasite in the insect host. Dutton accidentally inoculated himself through a wound on the hand at an autopsy and developed the disease which caused his death.

The particular tick in this case is the *Ornithodoros savignyi*. The prevention of the disease depends entirely upon a knowledge of the biology of the tick and efforts to guard against its bite, to prevent infection of the ticks, and to destroy them, as far as possible, in the infected regions.

LICE

The insects known as pediculi or lice are parasitic during their entire life on warm-blooded animals, including man. They are degraded, flat, rather elongate, wingless insects with a small head and stout legs which end in a strong claw, opposable to a projection at the tip of the penultimate joint. The mouth parts are of a very peculiar nature. There is a short beak or proboscis in front. Through this beak extends a slender stylet which has three parts. The stylet is used to pierce the skin of the host and the blood is thus sucked up through the proboscis. Lice usually walk sideways, but do not travel much and keep fairly close to one host. The eggs are slightly elongated and fastened to the hair of the host or clothing. They hatch in about ten to fifteen days, the young coming out of the top of the egg. These young do not differ much in structure from the adults, but are paler in color. They molt their skin a few times, probably four, before they reach the matured condition. The males are less numerous than the females, and ordinarily smaller. There are several generations each year, dependent, doubtless, on the temperature, but the life history is not thoroughly known for any species.

It is the blood-sucking habits of lice which render them dangerous parasites and capable of transmitting disease from one host to another. Fortunately, they do not readily change hosts, so that they cannot be considered quite as dangerous as some more active parasites. There are about 50 or 60 known species which are arranged in 15 genera and 4 families. It is *Pediculus vestimenti*, the clothes or body louse, which is mainly responsible for the transmission of typhus fever.

Three species of lice are found upon man: (1) *Pediculus capitis* (now *humanus*), the ova of which are attached to the hairs and can readily be seen as white specks, known as nits. (2) *Pediculus vestimenti* (or *corporis*), the clothes or body louse, lives on the clothing, and in sucking the blood causes minute hemorrhagic specks, commonly about the neck, back, and abdomen. (3) *Pediculus* (or *Phthirius*) *pubis* or crab louse is found in the parts of the body covered with short hairs, as the pubes; more rarely the axilla and eyebrows.

The prevention of lousiness is almost entirely a matter of personal cleanliness. However, the most scrupulous individuals may become infested. Lice may be passed directly from one person to another, or occasionally may be carried by flies, or other means. Beds in hotels and sleeping cars are sources of infection.

Human lice may be destroyed with kerosene, turpentine, carbolic acid (1-50), bichlorid of mercury solutions, tincture of cocculus indicus, and other well-known insecticides. It is comparatively easy to destroy the adult insect, but the eggs are resistant. On badly infected heads, therefore, the hair should be cut short. To free the hair of lice a good practice is to use equal parts of kerosene and olive oil. Rub the mixture well into the scalp, then cover the hair with a piece of muslin and fasten it about the head. Care must be exercised to avoid a lighted gas jet or flame. In the morning wash the scalp well with soap and hot water, then use a fine-toothed comb wet in vinegar to remove the nits. Repeat the treatment two or three nights.

In the case of the body louse the clothing should be boiled, baked, or steamed. Articles injured by heat may be subjected to sulphur fumes or dipped in carbolic acid solution. Carbon bisulphid and hydrocyanic acid are also effective.

For pubic lice white precipitate or mercurial ointment should be used and the parts thoroughly washed two or three times a day with soft soap and water.

The principal disease known to be transmitted by lice is typhus fever, but it is suspected in relapsing fever and other infections.

TYPHUS FEVER

Typhus fever was formerly confused with typhoid fever, but Gerhard in 1829 was the first to insist upon the non-identity of these two diseases. Previous to that time typhus fever was the prominent and prevailing disease, while typhoid fever was of secondary interest. Now the situation is reversed; typhoid fever has become pandemic, while typhus fever has receded with civilization and improvements in sanitation. Epidemics of typhus fever are now rare, except in a few places, notably the Grand Plateau of Mexico, where the disease prevails extensively and with a high mortality. It prevails also in certain portions of Ireland, in some provinces of France, portions of Russia, particularly Poland and the east sea provinces, and at times in Tunis, Algiers, and Egypt in Northern Africa; in Spain, Hungary, and certain provinces of the Baltic States.

Typhus fever last prevailed in epidemic form in the United States in New York in 1881-82 and again in 1892-93, and in Philadelphia in 1883. Since then, except for a few sporadic cases at our seaports, the

disease has been thought to be non-existent in the United States. However, Anderson and Goldberger[1] have recently shown that the symptom-complex known as "Brill's disease" is in reality typhus fever, and that the typhus fever of Europe and the typhus fever or "tabardillo" of Mexico are the same disease. It is now evident that typhus fever has been existent in New York a great many years, certainly since 1896, when Brill first observed cases of what was known, previous to the work of Anderson and Goldberger, as "Brill's disease." The disease in New York is generally mild, but seems to be on the increase; therefore, we face a new sanitary problem in this country.

Typhus fever, when prevalent in epidemic form, has been said by the older writers to be one of the most highly contagious of febrile diseases, doctors and nurses and others in close contact with the disease being almost invariably stricken. The sad case of Ricketts, who lost his life in endeavoring to unravel this pathological puzzle in Mexico, is still fresh in mind.

The period of incubation of typhus fever is from five to twenty days, with an average of twelve. One attack apparently confers a very definite immunity, second attacks being very unusual. The cause of the infection is unknown.

Methods of prevention have been given a sound foundation through the recent work of Nicolle of France, Ricketts and Wilder of the University of Chicago, and of Anderson and Goldberger of the U. S. Public Health and Marine Hospital Service. It is now clear that the virus exists in the circulating blood during at least all of the febrile stage and possibly in some instances for thirty-six hours after the crisis.

The disease may be transmitted by blood inoculations to chimpanzees and probably to all the lower monkeys. The virus as it exists in the circulating blood is apparently held back by the Berkefeld filter. It is not killed by freezing for eight days, but is deprived of virulence by heating at 55° C. for 15 minutes.

Monkeys that recover from the experimental disease show a definite immunity to subsequent infection.

Nicolle in 1909 was the first to report the transmission of typhus fever by the bite of the body louse (*Pediculus vestimenti*). Since then his work has been confirmed by Ricketts and Wilder and by Anderson and Goldberger. These latter authors have recently shown that the head louse (*Pediculus capitis*) may also transmit the infection. The rôle of the body louse in the transmission of typhus fever will receive ready support from students of the epidemiology of typhus fever, for this disease presents all the characteristics of insect-borne

[1] Anderson, John F., and Goldberger, Joseph: "The relation of so-called Brill's disease to typhus fever; an experimental demonstration of their identity." *Public Health Reports,* XXVII, February 2, 1912.

disease. Since the transmission of the disease by the body louse has been shown, we can understand why typhus fever prevails in epidemic form only in overcrowded, filthy, unhygienic surroundings, and the truth is readily understood of the oft-quoted sentence of Hirsch, that "the history of typhus is the history of human wretchedness."

The disease has greatly decreased from civilized centers with diminution in lousiness. The prevention of typhus now focuses itself upon the eradication of the body louse. Fortunately, this insect does not of itself travel far, but it may be carried many miles upon the body or in the clothing. The eradication of the body louse is largely a question of personal cleanliness, and, so far as typhus fever is concerned, is closely interwoven with squalor, ignorance, and poverty.

Now that we know how the disease is spread, measures may be intelligently applied for its prevention, these measures being primarily directed to the destruction of the *Pediculus vestimenti* and its eggs. When a case of typhus fever is discovered the patient should be removed to a vermin-free room or hospital. The patient's clothes should be removed and either placed in boiling water or a 1-500 solution of bichlorid of mercury for the destruction of lice and their eggs. The patient's hair should be clipped and he should then be given a thorough sponging with a 1-2,000 solution of bichlorid of mercury for the destruction of lice eggs. The room or apartment from which the patient was removed should be thoroughly fumigated by the burning of sulphur for the destruction of lice, and the room kept sealed for at least 12 hours.

The fact should be kept constantly in mind that the louse is necessary for the spread of typhus fever, just as the mosquito is for the spread of malaria, and our efforts toward prophylaxis should be conducted with this point continuously in mind. Even with the knowledge of the mode of transmission of typhus fever individual prophylaxis is still somewhat difficult, especially where infected insects abound in thickly populated centers.

Those whose duties—such as doctors and nurses—take them into an infected area should avoid outer clothing which is liable to brush against the furniture, bedding, etc. The skirts of nurses should be sufficiently short to avoid touching the floor; trousers should be rolled above the shoe-tops and the sleeves above the elbows, so that occasional vermin which may lodge on the hand may be more readily detected. Eucalyptus oil has been recommended for smearing the neck, wrists, and ankles. Personal prophylaxis may also be assisted through the use of gloves, veils, netting, and similar mechanical devices. The clothing worn by those attending cases of the disease where lice are present should be frequently changed and close attention given to personal cleanliness.

BEDBUGS

Cimex lectularius has been carried by man to all parts of the inhabited world. It has become a true domesticated animal and has accommodated itself well to the environment of human habitations. The bedbug has no wings and a very flat body, which enables it to hide in the narrowest chinks and cracks of beds and wells. It may subsist for incredibly long periods of time without food. It is nocturnal in its habits.

The pronounced odor of this insect is produced by certain glands opening on the back of the abdomen in young bugs and on the under

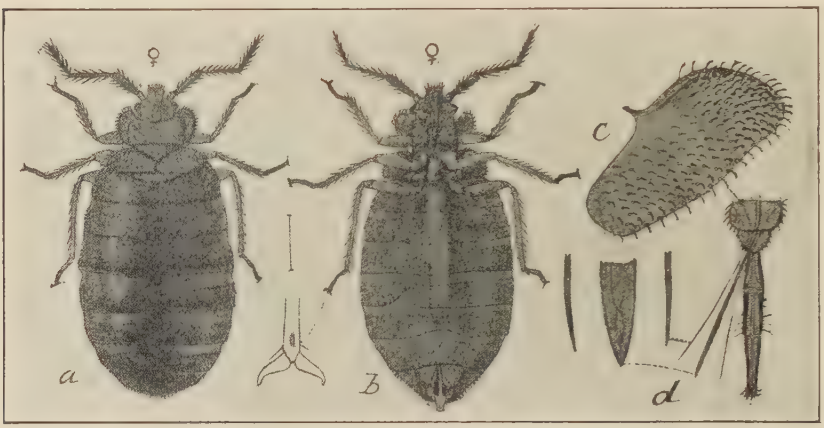

FIG. 46.—THE BEDBUG.
a, Adult female, gorged with blood; *b*, Same from below; *c*, Rudimentary wing pad; *d*, Mouth parts. (Marlatt.)

side of the metasternum in the adults. The odor is common to most members of the group to which this insect belongs. It is useful in plant bugs, protecting them from their enemies.

The bedbug undergoes an incomplete metamorphosis, the young being very similar to their parents in appearance, structure, and habits. The eggs are white, oval objects having a little projecting rim around one edge, and are laid in batches of from six to fifty, in cracks and crevices where the bugs go for concealment. The eggs hatch in a week or ten days and the young escape by pushing the lid within the projecting rim from the shell. At first the larvæ are yellowish-white, nearly transparent, the brown color of the more mature insect increasing with the later molts. During the course of development the skin is shed five times, and with the last molt the minute wing pads, characteristic of the adult insect, make their appearance. Marlatt found that under favorable conditions about seven weeks elapse from the egg

to the adult insect, and that the time between each molt averages about eight days. Without food they may remain unchanged for an indefinite time. Ordinarily but one meal is taken between molts, so that each bedbug must puncture its host five times before becoming mature, and at least once afterward before it can develop eggs.

The presence of bedbugs in a house is not necessarily an indication of neglect or carelessness. They are very apt to get into trunks and satchels of travelers or may be introduced in the homes upon the clothing of servants, workmen, or visitors. The bedbug is quite capable of migrating from one house to another. Ships are almost sure to be infested with them. They are not specially limited by cold, and are known to occur well north. They thrive particularly in old houses which are full of cracks and crevices, in which they can conceal themselves beyond easy reach. The biting organ of the bedbug is similar to that of other Hemipterous insects. The skin of the host or victim is pierced with four thread-like hard filaments or setæ, which glide over each other with an alternating motion and thus pierce the skin. The blood is drawn up through the beak, which is closely applied to the point of puncture. The bite of the bedbug is decidedly poisonous to some individuals, resulting in a swelling and disagreeable inflammation.

The Suppression of Bedbugs.—On account of its habits of concealment the bedbug is usually beyond the reach of the ordinary insect powders, which are practically of no avail against it. If iron or brass bedsteads are used, the eradication of the insect is made easier. Large wooden bedsteads furnish many cracks and crevices into which the bugs can force their flat thin bodies, and extermination becomes a matter of considerable difficulty. The most practical way of eradicating bedbugs is by a very liberal application of gasolene, benzine, kerosene, or any other of the petroleum oils. These must be introduced into all crevices with small brushes or feathers, or by injecting with small syringes; a saturated solution of corrosive sublimate in water is also of value, and oil of turpentine may be used in the same way. The liberal use of scalding hot water or soap suds wherever it may be employed without damage to furniture is also an effectual method of destroying both eggs and active bugs. Fumigation with hydrocyanic acid gas, sulphur dioxid, or carbon bisulphid are alike effective. Crevices in warm parts of the room are favorite nesting places, as under picture mouldings, or over door frames.

In sleeping cars and other places where hydrocyanic acid gas may be used without fear of accidents, this is the most efficacious and least destructive method.

The bedbug has long been under suspicion as an intermediate host in the transference of many communicable infections. There is more

than a suspicion that it is concerned in relapsing fever, in kala-azar, and it has been accused of carrying the bacteria of tuberculosis, leprosy, and many other diseases.

KALA-AZAR

Kala-azar is a tropical infection characterized by anemia and enlargement of the spleen. It is caused by a parasite which occurs in great numbers in the spleen and which, upon culture media, develops into a flagellated organism resembling the trypanosomes. The trypanosomes were discovered by Leishman and Donovan in the spleen and liver and the epithelium of the blood vessels. Manson and Low found similar bodies in the ulcerous mucous membranes of the intestines, and Marchand and Ledingham found the same peculiar bodies in the cells of the bone marrow and lymphatic glands. Rogers cultivated the parasites from the spleen of patients suffering with kala-azar upon agar streaked with fresh human blood. Flagellate forms developed. This was confirmed by Christophers, who used Novy's method of growing trypanosomes upon the water of condensation of blood agar tubes. The kala-azar parasites grown in artificial culture media have a cilium but no membrane.

References.—The literature upon insects and insect-borne diseases is very widely distributed. Many of the entomological facts contained in this chapter have been taken from "The Insect Book" by L. O. Howard and the many excellent publications of Howard and his colleagues of the Bureau of Entomology, Department of Agriculture. The Government publications may be had upon application to the Superintendent of Documents, Washington, D. C. Many of the facts concerning the prevention and destruction of mosquitoes have been taken from articles in the *Public Health Reports* of the Public Health and Marine Hospital Service. In the chapter upon insecticides free reference has been made to my own book upon "Disinfection and Disinfectants," as well as my other writings and unpublished work in different phases of this subject.

CHAPTER V

MISCELLANEOUS DISEASES

INFANTILE PARALYSIS

(*Acute Anterior Poliomyelitis*)

An entirely new literature upon the subject of infantile paralysis is now being constructed. The chief contributors to this recent advance in our knowledge have been Wickman of Sweden, who, in 1905-06, gave us a new symptomatology, and defined clinical types not before recognized. Wickman made the first systematic study of the disease from an epidemiological point of view, and found evidence that it was contagious, though usually slightly so. He directed especial attention to several factors in its spread, viz.: routes of travel, public gatherings of children, abortive or ambulant cases, and healthy intermediate carriers. In the spring of 1909 Landsteiner and Popper succeeded in transmitting the disease to two monkeys by inoculating them with the spinal cord of a child who had died of infantile paralysis. Later in the year Flexner and Lewis obtained the same results, and further transmitted the infection from monkey to monkey through an indefinite number of passages. To Harwitz and Scheele of Norway we are indebted for formulating the pathologic anatomy of the affection.

Infantile paralysis is now properly regarded as a communicable disease. The virus is filterable, that is, "ultramicroscopic," yet coccæ forms have been described by Noguchi and Flexner in artificial cultures. (*J. A. M. A.*, Feb. 1, 1913, LX, 5, 362.)

It appears that infantile paralysis is becoming more and more common and more widespread of late years. This increase cannot be accounted for by the fact that the disease is now better known and more readily recognized. Bergenholtz, in 1881, described the first outbreak with sufficient accuracy to accept infantile paralysis as a new disease. Since that time the number of outbreaks and the number of cases have progressively increased, as shown in the following table:

	Cases	Outbreaks	Av. No. of Cases per Outbreak
1880-1884.....	23	2	11.5
1885-1889.....	93	7	13.
1890-1894.....	151	4	38.
1895-1899.....	345	23	15.
1900-1904.....	349	9	39.
1905-1909.....	8,054	25	322.

Recent outbreaks have occurred in Norway and Sweden, Austria, Germany, Holland, England, Spain, France, the United States, and Cuba. Of the 8,054 cases reported in 5 years (1905-09), the United States contributed 5,514 cases or about five-sevenths of the total number.

Epidemics of poliomyelitis have prevailed in all quarters of the world. The disease has been most prevalent in the northern parts of Europe and of the United States. Epidemics have been more severe, and the case rates have been higher, in small towns and rural districts than in the more densely populated cities. Even in the cities the disease does not especially strike the crowded districts. Cold countries having marked seasonal variations in temperature have been most affected, but the disease is always most prevalent in the warm, dry months, from May to November in the northern hemisphere and November to May in the southern hemisphere. Sporadic cases may occur at any time throughout the year. The great majority of cases occur in children under five years of age. From the standpoint of prevention it is important to note that social and hygienic conditions apparently have no influence whatever in determining the infection. All classes are affected in about equal proportion.

The virus of the disease is present in greatest virulence or concentration in the spinal cord of infected persons and animals. One one-hundredth of a cubic centimeter of an emulsion of cord, or less, is sufficient to infect a monkey. The virus is also quite constantly present in the brain and other organs and tissues, as, for instance, the mucous membrane of the nose and pharynx, the mesenteric glands, the axillary and inguinal lymph nodes, also in the blood, and in the cerebrospinal fluid. The virus has been demonstrated in the feces. The suspicion that the alvine discharges may, therefore, be virulent is sufficient indication that they should be disinfected in all cases until further knowledge of the subject is at hand.

The experimental disease in monkeys may be produced with certainty by injecting the virus directly into the central nervous system, preferably the brain. Monkeys may also be infected by introducing the virus subcutaneously or into the peritoneal cavity, and even by intravenous inoculation. They have been infected by placing virulent

material upon the healthy mucous membrane of the nose and also by inhalation of the infectious material forced into the trachea, and finally by introducing the virus into the stomach, along with an opiate, to restrain peristalsis. Leiner and Weisner have infected monkeys through the uninjured nasal mucous membrane. This, however, is an uncertain method of inoculation. Monkeys have so far never been known to contract the disease spontaneously, even though they are kept in intimate association with infected monkeys. There are many similar paralytic diseases of the lower animals, but, so far as known, infantile paralysis as a natural infection is peculiar to man. Recently Rosenau and Brues, and also Anderson and Frost, have transmitted the disease from monkey to monkey through the bite of the stable fly.

Resistance of the Virus.—The virus of anterior poliomyelitis is killed by a temperature of 45° to 50° C. in half an hour; also by comparatively weak disinfectants, such as a 1-500 solution of permanganate of potash, 1 per cent. menthol in oil, a powder containing menthol, 0.5 per cent., salol, 5 per cent., boric acid, 20 per cent. (Landsteiner and Levaditi), and a dilution of perhydrol (Merck) equivalent to 1 per cent. of peroxid of hydrogen. The virus is not destroyed by very low temperatures nor by drying over caustic potash, or *in vacuo* for a considerable period. A virulent cord has been kept for almost 5 months in pure glycerin without losing its virulence, resembling in this respect rabies, vaccine, and other filterable viruses, and differing for the most part from non-spore-bearing pathogenic bacteria which are usually killed by pure glycerin in a short while.

Immunity.—One attack of infantile paralysis apparently confers a high degree of immunity. Recurrent cases and second attacks have been reported. Monkeys which have recovered from the infection show a high degree of resistance, in that they are not susceptible to infection by again inoculating them, and their blood serum contains antibodies capable of rendering the virus harmless. That is, if the blood serum of an immune monkey is mixed with an emulsion of virulent spinal cord and the mixture allowed to stand for several hours, the virus is no longer capable of producing the infection in susceptible animals. This property has been used by Anderson and Frost to corroborate the clinical diagnosis in abortive cases. The blood of a person who has not had the disease does not neutralize the virus; therefore, if the injection of the virus previously treated with human serum fails to produce the infection in susceptible monkeys, it may be taken as evidence that the serum contained specific antibodies and came from an individual who has had the disease.

Modes of Transmission.—CONTACT THEORY (BASED UPON THE ASSUMPTION THAT THE VIRUS IS DISCHARGED FROM THE MOUTH AND NOSE AND ENTERS THROUGH THE SAME CHANNEL).—There is evidence to sup-

port the theory that the disease is directly transmissible from person to person and there is a suspicion that healthy carriers play an important rôle in spreading the infection. This view was enunciated by Wickman and received support through the experiments of Kling, Pettersson and Wernstedt, and also Flexner. It is known that the mucous membrane of the nose and throat contains the virus, and in one case the salivary glands were shown to be infective. Osgood and Lucas demonstrated that the nasal mucous membrane of two monkeys experimentally inoculated with poliomyelitis remained infective for 6 weeks in one case and 5½ months in another. This very important observation strengthens the suspicion of the existence of chronic human carriers. If healthy carriers continue to spread the infection months after the attack, it increases the difficulty of suppressing the disease, and further renders doubtful the efficiency of strict isolation and prophylactic measures directed only to persons in the acute stage of the disease. The fact that the mucous membrane contains the virus is not, however, sufficient proof that the virus is liberated and discharged in sufficient amount in the secretions from the mouth and nose to be a menace. In a series of 18 cases Rosenau, Sheppard and Amoss [1] were unable to demonstrate the virus in the nasal and buccal secretions obtained from persons in various stages of convalescence. Strauss [2] had similar negative results in a series of 10 cases. On the other hand, Kling, Pettersson and Wernstedt [3] report successful results; by experiments upon monkeys they demonstrated the infectiousness of buccal and intestinal secretions of living subjects. Flexner has recently also reported one successful attempt in demonstrating the virus in the buccal secretions.

The Insect-borne Theory.—Infantile paralysis shows no tendency to prevail in congested centers or to spread in hospitals, schools, institutions, and other crowded places; its seasonal prevalence corresponds to the seasonal prevalence of most insects, and does not correspond to the seasonal prevalence of diseases spread through secretions of the mouth and nose, such as diphtheria, scarlet fever, smallpox, etc. Many other factors, brought to light by the studies of the State Board of Health of Massachusetts upon the epidemiology of the disease, under the able direction of Dr. Mark Richardson, indicate that the disease is not a contagious one. These studies [4] gradually focused attention upon some

[1] Rosenau, M. J., Sheppard, P. A. E., Amoss, H. L., *Boston Med. and Surg. Jour.*, May 25, 1911, CLXIV, 21, pp. 743-748.

[2] Strauss, I., *J. A. M. A.*, April 22, 1911, LVI, 16, 1192.

[3] Kling, C., Pettersson, A., and Wernstedt, W., Report from the State Medical Institute of Sweden to the XV International Congress on Hygiene and Demography, Washington, D. C., 1912. Also, *Zeitschr. f. Immunitätsforch. u. exper. Therapie*, Bd. XII, Jena, 1912.

[4] Richardson, M. W., *Monthly Bull.*, State Board of Health of Mass., Sept., 1912, 7, 9, pp. 308-315.

Lovett, R. W., Report to the Mass. State Board of Health, 1907.

Report to the Mass. State Board of Health, 1908, 1909, 1910, 1911.

insect, the stable fly (*Stomoxys calcitrans*) in particular. Rosenau and Brues [1] demonstrated that the virus may be transmitted from monkey to monkey through the bite of the stable fly. These results were soon confirmed by Anderson and Frost.[2] The insect-borne theory seems to fit the case as the disease is known in Massachusetts. It will, however, require much additional study to determine what rôle *Stomoxys calcitrans* plays in spreading the infection in nature.

Other Theories.—It has been suggested that the virus may be air-borne in the sense that it is carried in the dust. Neustaedter and Thro [3] have infected monkeys from dust collected from sick rooms. Infected food, or transmission through wounds and other means, have not been ruled out of consideration.

Prevention.—No definite or effective system of prevention can be formulated until we are sure of the mode of transmission. Meanwhile health authorities are entirely justified in requiring cases to be reported, isolated, and all known lines of preventive measures applied, such as disinfection, screening, and guarding against insects, allaying unnecessary dust, etc. A fly campaign directed with especial reference to the stable fly is plainly indicated, and the infection must also be fought as one conveyed from man to man directly. Until the modes of transmission of the disease are established, however, we can have no confidence in our prophylactic measures, which most resemble the old "shotgun" prescription.

The following measures are recommended: The patient should be isolated as completely as possible in a clean, bare room, well screened to keep out insects. This is a good practice despite the fact that the disease shows no tendency to spread in children's asylums, hospitals, and other institutions, or even in the home. The same statement, however, was made of typhoid fever not many years ago. Visiting should be interdicted and only the necessary attendant should be allowed to come in contact with the patient. All discharges, including sputum, nasal secretions, urine, and feces, should be thoroughly disinfected, and special care should be taken that cups, spoons, remnants of food, etc., which may have become contaminated by the patient are burned, scalded, or otherwise purified.

Towels, bed linen, and other fabrics should be boiled or dipped into a germicidal solution strong enough to destroy the typhoid bacillus. The nurse and physician should observe the same precautions re-

[1] Rosenau, M. J., and Brues, C. T., *Monthly Bull.*, State Board of Health of Mass., Sept., 1912, 7, 9, pp. 314-318. Also Brues and Sheppard, *Jour. of Econom. Entomology*, Aug., 1912, V, 4, 305.

[2] Anderson, J. F., and Frost, W. H., *Pub. Health Reports*, Oct. 25, 1912, XXVII, 43, pp. 1733-1736.

[3] Neustaedter, M., and Thro, W. C., *N. Y. Med. Jour.*, Sept. 23, 1911, XCIV, 13.

garding their hands and clothing as are recommended in attending a case of scarlet fever.

The period during which the isolation should be maintained cannot even be guessed at. Children are usually not permitted to return to school for at least three weeks, but, if chronic carriers play the important rôle now suspected, this time would be far too short in many instances.

Since the virus can be killed experimentally by a 1 per cent. solution of peroxid of hydrogen, antiseptic gargles, sprays, and nose washes of this solution are recommended to be used by the patient, the nurse, and physician, and other members of the family. In the presence of an epidemic, street and house dust should be kept down by sprinkling, oiling, and the other means employed for this purpose. Dust should be allayed whether there is an epidemic of infantile paralysis or not. During epidemics children should be kept away from public gatherings, prohibited from using public drinking cups, and special attention given to the diet to prevent gastrointestinal disorders, for many a case of infantile paralysis starts with a digestive upset.

CHICKENPOX

Chickenpox is one of the minor communicable diseases, in that the mortality is practically nil and that complications and sequelæ are rare. Chickenpox is very readily communicable and spreads through families or institutions, but does not occur in widespread epidemics. The cause of the disease and its modes of transmission are not known. The virus is not contained in the content of the vesicle. Tyzzer and others made numerous inoculations with both clear and clouded vesicle contents without results. The disease is probably peculiar to man; animal inoculations have so far proven negative. The period of incubation is probably from fourteen to sixteen days, and one attack produces a definite immunity. No age is exempt.

The differential diagnosis between chickenpox and smallpox is often an important and difficult public health matter. The distinction may be made by introducing some of the contents of the vesicle into the skin of a well-vaccinated person. If chickenpox, an immediate reaction results; if smallpox, no reaction results. Monkeys are not susceptible to chickenpox but may be given smallpox. The differential diagnosis may also be made from the presence of vaccine bodies in smallpox and their absence in chickenpox. These bodies are best demonstrated by introducing some of the virus upon the cornea of a rabbit, and examining the vesicles which form.

Health officers should require cases of chickenpox to be reported, if for no other reason than that it is often mistaken for smallpox. The

differential diagnosis may be made in doubtful cases by a histological examination of the pock, or by inoculating the contents of the vesicle upon the cornea of rabbits. In sections of the skin lesion the vaccine bodies are found in smallpox, not in chickenpox; the vesicle of the former is multilocular, the latter unilocular. The vesicle upon the cornea of rabbits produced by smallpox is distinct and contains the vaccine bodies; the lesion resulting from chickenpox is trifling and does not contain the vaccine bodies.

The prevention of chickenpox depends upon isolation and disinfection at the bedside. Children with chickenpox should not be permitted to go to school.

GLANDERS

Glanders or farcy is a widespread communicable disease of horses, mules, asses, and other animals, and is readily communicated to man. In both man and horses it is remarkable for its fatality. The disease is characterized by the formation of inflammatory nodules either in the mucous membrane of the nose (glanders) or in the skin (farcy).

Glanders is caused by the *Bacillus mallei,* which corresponds to the spore-free bacteria so far as its resistance is concerned. In general the bacillus of glanders is killed by the same agents used against the tubercle bacillus, which it resembles in some particulars.

The infection may be introduced into the system either through the skin or mucous membrane, and is usually communicated directly from the horse to man by contact with the infected discharges. The disease is sometimes communicated from man to man. Washerwomen have become infected from the clothes of a patient.

The bacillus of glanders does not have a spore. It is comparatively frail and readily destroyed by the usual physical and chemical germicidal agencies used against spore-free bacteria. The bacillus, however, is frequently protected by albuminous matter or buried in the dirt of stables, water troughs, harnesses, and other objects. While the naked germs of glanders are readily destroyed, they are frequently hard to get at; cleanliness is, therefore, imperative.

The prevention of glanders in man depends primarily upon the suppression of the disease in horses. The only difficulty in controlling the disease in horses lies in the early diagnosis and recognition of mild or missed cases, which are very common. Horses affected with occult or latent glanders are important factors in the propagation of the infection. The clinical diagnosis in the frank cases is made without difficulty from the characteristic symptoms and the lesions, but laboratory aid is necessary to discover the mild cases.

Diagnosis.—The diagnosis of glanders may be made by: (1) the mallein test; (2) the agglutination test; (3) the Strauss reaction;

(4) isolation of *B. mallei* in pure culture; and (5) complement fixation. All these tests serve a definite purpose. However, the mallein test, the agglutination test, and the Strauss reaction are not sufficiently reliable to be entirely satisfactory. The isolation of the glanders bacillus in pure culture is definite and final, but time-consuming. The diagnosis of glanders by complement fixation is at present our most reliable, most satisfactory, and quickest method of recognizing the disease.

The Mallein Test.—Mallein is a product of the glanders bacillus corresponding to tuberculin. The injection of mallein into normal animals produces no reaction, whereas the injection into glanderous animals causes a rise in temperature and a local reaction about the lesions. With the mallein test a large proportion of latent and occult cases of glanders can be diagnosed, but the test must be made and interpreted by an experienced veterinarian, else the results may be unreliable. The mallein test fails to give a typical reaction in a considerable number of glanderous animals; on the other hand, a reaction may follow the injection of mallein in the absence of glanders. Thus mallein is not an entirely reliable diagnostic agent and has never been considered as specific in the detection of this disease as tuberculin for the diagnosis of tuberculosis.

The Agglutination Test.—The agglutination test is of value in all cases of recent infection, the blood serum possessing a very high agglutinating power—1-1,000 and higher. In chronic glanders the agglutinating power of the blood may be very low—1-400 or less; in some cases even lower than that of normal blood serum—which may be 1-800 and even higher. It is, therefore, plain that agglutination tests alone do not constitute an entirely satisfactory diagnostic method for glanders. It may be used as an adjunct to other tests.

The Strauss Reaction.—The Strauss[1] reaction for the diagnosis of glanders consists in inoculating male guinea pigs into the peritoneal cavity with material containing virulent *B. mallei*, which causes an enlargement of the testicles. A positive reaction associated with organisms resembling those of glanders, and typical cultures obtained from the lesions, are unfailing evidence of the presence of the specific virus. Failure to obtain the reaction is not proof that a suspected specimen may not have come from a horse or animal with glanders. Arms[2] recommends that it is better to use more than one guinea pig in testing suspected material, and that, before inoculating, it is well to make a microscopic examination as a guide to the dosage. A culture made from the swab often aids in the early diagnosis. Guinea pigs should be kept under observation for a month, and if a

[1] *Compt. Rend. Acad. d. Sc.*, 1889, CVIII, p. 530.
[2] *J. A. M. A.*, LV, 7, Aug. 13, 1910, p. 591.

lesion of any kind is present an autopsy should be made and cultures taken.

The Isolation of B. Mallei in Pure Culture.—The bacillus of glanders may be isolated by introducing some of the suspected material subcutaneously and also intraperitoneally into male guinea pigs. In this way pure cultures may be obtained from the pus or necrotic foci in the spleen, which follow subcutaneous inoculation; or from the characteristic enlargement of the testicle which is observed in animals inoculated intraperitoneally. The organism isolated must be studied for cultural, morphological, and biological characters. The isolation of the bacillus in pure culture gives positive information of unquestioned character in any critical case. The method is not generally applicable to the diagnosis of glanders because it requires too much time and may occasionally fail to discover the bacillus.

Complement Fixation.—In 1909 Schütz and Schubert[1] published the results of their important work on the application of the method of complement fixation for the diagnosis of glanders. The splendid results obtained constitute, without doubt, the most reliable method for the diagnosis of glanders which we have at our command at the present time. The complement fixation test is, in fact, one of the most specific of the biological tests in immunity. It is readily applicable to the case of glanders. The essential elements used in the test are as follows:

The *hemolytic system* consists of the washed red blood corpuscles of a sheep and the blood serum of a rabbit which has been injected with the washed red blood corpuscles of a sheep. Strong, vigorous rabbits are selected and three injections of the sheep's corpuscles are made at intervals of 7 days, using 7 c. c., 10 c. c., and 12 c. c. of the red corpuscles of the sheep suspended in like amounts of isotonic salt solution. The blood serum of a rabbit so treated contains the hemolytic amboceptors. The rabbit's blood serum is heated to 56° C. for half an hour in order to destroy the complement. The titer, or amount of amboceptor contained in the rabbit serum, must be determined. The hemolytic system, then, consists of rabbit serum containing amboceptor, plus washed red blood corpuscles of the sheep.

Complement.—The complement is contained in the fresh blood serum of a healthy guinea pig. The blood serum of the guinea pig should be titrated in order to determine the amount of complement present. It is always necessary to determine the smallest quantity of complement to be used for the final test.

Antigen.—The antigen consists in an extract obtained by shaking

[1] Schütz and Schubert: "Die Ermittelung der Rotzkrankheit mit Hilfe der Komplementablenkungsmethode." *Archiv für wissenschaftliche und praktische Tierheilkunde.* Bd. 35, Heft 1 and 2, pp. 44-83, 1909.

glanders bacilli in salt solution. The bacillus is grown in pure culture on 2 per cent. acid glycerin agar. A luxuriant growth upon the surface of the medium is usually obtained in 48 hours. This is suspended in salt solution, heated to 60° C. for four hours in order to kill the bacilli. After heating the dead bacilli are shaken in the salt solution in a special apparatus for four days. The bacilli are separated in the centrifuge and the clear supernatant liquid is drawn off and preserved with carbolic acid. The strength of this extract must be determined by suitable methods of titration.

Technique.—The test is carried out by adding together, in proper proportions, the following: (1) The blood serum of the horse to be tested; (2) the antigen (extract of glanders bacilli); (3) complement (fresh guinea pig serum); and (4) the hemolytic system. If the blood serum of the horse to be tested contains the specific amboceptors these will unite with the bacteria, fix the complement, and thus prevent hemolysis. If the blood serum of the horse to be tested does not contain these specific amboceptors, this fixation of the complement cannot take place and hemolysis results. Therefore, the absence of hemolysis means the presence of glanders, and vice versa. The tests must always be carried out with controls and carefully conducted as to the amount of each substance used, the temperature and time.[1]

Prevention.—When glanders is discovered or suspected among horses in a stable, the blood of all the horses in the infected stable should be drawn and tested in the manner above described. All animals whose serum shows complement fixation should be destroyed without further consideration. After the animals have been killed and properly disposed of, the stable should be thoroughly cleansed and disinfected. All other horses which have in any way been associated with the infected animals should be carefully watched and tested again after three weeks, and, should there be no indication of the disease in the second test, the stable may be considered free from the infection; otherwise the infected animals should be destroyed and the tests repeated every three weeks until the disease has been eliminated.

The eradication of glanders from a stable often means considerable loss and sometimes a sacrifice of valuable animals, but it is only through vigorous measures that the disease may be controlled. In the disinfection and cleansing special attention should be paid to the stalls, harnesses, water troughs, bits, food containers, curry combs, sponges, and other objects exposed to the infection, which is eliminated mostly in the secretions from the mouth and nose. The common drinking trough for horses spreads the infection.

[1] A complete description of the diagnosis of glanders by complement fixation, giving in full all the details, will be found in *Bull. 136*, Bureau of Animal Industry, Apr. 7, 1911, by Mohler and Eichhorn.

The personal prophylaxis of glanders in man depends upon the education and care of those who have to handle horses. In working with horses known to be infected rubber gloves, disinfection, and other methods of protection should be employed. Special care should be taken to prevent the spread of the infection through the discharges or by infected fomites from human cases. Fatal accidents have occurred in laboratories in research workers handling pure cultures of *B. mallei.*

ANTHRAX

Anthrax belongs to that group of diseases which occurs primarily in the lower animals and secondarily in man. The infection is found in horses, cattle, sheep, and other cloven-hoofed animals, and may be transmitted experimentally to mice, guinea pigs, rats, and rabbits. Cold-blooded animals and birds, as well as dogs and swine, are refractory.

In man the infection may enter the skin (malignant pustule) or may enter the lungs (wool sorters' disease), or may enter the digestive tract and produce intestinal lesions. In anthrax of the skin the infection usually enters through slight abrasions, scratches, or small wounds, especially on the forearm, hand, or face. Most of the cases occur in butchers or persons who have to handle infected carcasses. The spores have been carried to the skin by flies and may be inoculated by the bite of the stable fly.

Wool sorters' disease, or anthrax of the lungs, appears to be due to the inhalation of anthrax spores. It is observed only among persons who handle skins or who work with horse hair or other raw materials from animals afflicted with anthrax.

The mode of transmission in intestinal anthrax is through meat from an anthrax cadaver which is partaken of without proper cooking.

Schuberg and Kuhn [1] have shown that anthrax may be transferred from animal to animal through the bite of the stable fly (*Stomoxys calcitrans*).

Resistance.—The anthrax spore is exceedingly resistant to heat and external influences, such as dryness and sunlight, and also to germicidal agents. Its resistance may be compared to the tetanus spore page 70.

Immunity.—A number of species of animals have a natural immunity to anthrax, and an artificially acquired immunity may be induced in cattle or sheep through the injection of attenuated cultures, in accordance with the classical method of Pasteur. These procedures are not applicable to man. The prevention of the disease in man must first be directed to a suppression of the disease in animals. The sick

[1] Arbeiten a. d. kaiserl. Ges.-Amt., Bd. XL, Heft 2, 1912.

animals should be isolated, or, better, killed, and the carcasses burned or buried at least three feet deep. The carcasses may be "tanked," that is, subjected to a prolonged exposure to steam under pressure. Tanks for this purpose are found in all the larger slaughter houses. It is important in handling the body of an animal dead of anthrax not to open it or shed blood, for the bacillus does not produce its spore except in the presence of oxygen, that is, the bacilli are mainly in the blood and internal organs and will not sporulate as long as access to the air is prevented.

Prevention.—The chief preventive measure so far as man is concerned is the disinfection of all raw material in those trades in which horse hair, hides, and other substances liable to harbor the anthrax spore are handled. Veterinary surgeons who conduct autopsies upon anthrax animals should exercise unusual precautions, and, if practicable, wear rubber gloves.

Ponder[1] recommends the following process to destroy anthrax infection in hides: The dry hides are placed for 24 hours in a "soak" which is made to contain 1 to 2 per cent. of formic acid and 0.02 per cent. of bichlorid of mercury, and then salting them with sodium chlorid. The action of the "soak" is to swell up the fibers of the hide by causing them to absorb water, the result being that the hide returns to a condition closely resembling that in which it was taken from the animal's carcass. This permits the bichlorid of mercury to permeate and exert its germicidal action.

FOOT-AND-MOUTH DISEASE

Foot-and-mouth disease is also known as aphthous fever, epizoötic catarrh, and eczema contagiosa. It is an acute and highly communicable disease, generally confined to cloven-footed animals, and characterized by an eruption of vesicles on the mucous membrane of the mouth and on the skin between the toes and above the hoofs. The vesicles rupture, forming erosions and ulcers. There are also salivation, tenderness of the affected parts, loss of appetite, lameness, emaciation, and diminution in the quantity of milk secreted.

Foot-and-mouth disease is primarily a disease of cattle and secondarily of man. Hogs, sheep, goats, buffalo, American bison, camel, chamois, llama, giraffe, antelope, and even horses, dogs, and cats may occasionally become infected.

The disease prevails in European countries and occasions great economic loss. The mortality is low; the serious losses depend chiefly upon the diminution of the milk secretion and the loss of flesh in the affected animals.

[1] *Lancet*, London, Nov. 4, CLXXXI, No. 4601, pp. 1247-1314.

Foot-and-mouth disease has appeared in the United States only on five different occasions—in 1870, 1880, 1884, 1902-3, and 1908. Every outbreak on American soil has thus far been followed by its complete suppression through the application of well-known preventive measures, such as isolation, destruction and burial of the affected herds, disinfection, and a systematic inspection of all farms in the infected area to detect cases of the disease.

Löffler and Froesch in 1898 showed that the virus will pass the finest porcelain filters. This was the first ultramicroscopic virus discovered. The specific principle is contained in the serum of the vesicles; in the saliva, milk, and various other secretions and excretions; also in the blood during the rise of temperature.

No definite immunity is rendered by an attack. The period of incubation is variable, usually from two to six days or longer, exceptional instances being prolonged to fifteen or even eighteen days.

The disease in man is a direct counterpart of that in cattle. The infection is transmitted to man through the ingestion of raw milk, buttermilk, butter, cheese, and whey from animals suffering with foot-and-mouth disease. It may also, though more rarely, be transmitted directly from the salivary secretions or other infected material which gains entrance through the mucous membrane of the mouth. It is doubtful whether the disease can be transmitted to man by cutaneous or subcutaneous inoculation, though it is probable that the infection may be communicated if the virus enters the blood directly through wounds of any kind. Children are most frequently infected by drinking unboiled milk during the time in which the disease is prevalent in the neighborhood, while persons in charge of diseased animals may become infected through contact with the affected parts or by milking, slaughtering, or caring for the animals. The disease is usually very mild in man; death practically never results, except in weakened children, and then from secondary complications.

The original experiments of Löffler and Froesch, as well as recent experiments which have been made in Denmark and Germany, indicate that the infection is destroyed comparatively readily by heat or the usual antiseptics. Milk pasteurized at a temperature of 60° C. for 20 minutes is safe, so far as infection from foot-and-mouth disease is concerned.

Foot-and-mouth disease has a special interest on account of the fact that it may be associated with vaccinia. The symbiosis between the infections of vaccinia and foot-and-mouth disease is remarkable. Animals vaccinated with the mixed virus, as a rule, show the lesions of only one of these diseases, namely, vaccinia. Nevertheless, the infectious principle of the other, foot-and-mouth disease, remains in the vaccinal eruption. Vaccine virus has been known to contain the in-

fection of foot-and-mouth disease.[1] Glycerin acts as a preservative for the virus of foot-and-mouth disease, so that it may remain viable in glycerinated vaccine virus a very long time. No instance of the transmission of foot-and-mouth disease to man through vaccine virus has been recorded, and it is doubtful, in view of the known facts, whether it is possible to reproduce the disease in man by the cutaneous inoculation commonly used in the process of vaccination. The prevention of foot-and-mouth infection in vaccine virus is assured through federal inspection and through special tests (see vaccine virus, page 20.

The prevention of foot-and-mouth disease consists (1) in a cattle quarantine, to keep it out of countries where it does not exist; (2) in the elimination of the disease in cattle through isolation of infected herds, destruction and burial of the sick animals, and disinfection; (3) the disease in man may be avoided by care in the selection of the animals from which milk is taken or by pasteurization of the milk when foot-and-mouth disease is prevalent.

MALTA FEVER

Malta fever is a general infection not unlike other specific bacteriemia, such as typhoid fever. It is caused by the *Micrococcus melitensis,* discovered by Bruce in 1887 during the earlier days of bacteriology. Clinically the disease is characterized by profuse perspiration, constipation, frequent relapses often accompanied by pains of a rheumatic or neuralgic character, and sometimes swelling of the joints or orchitis. Malta fever is further characterized by its low mortality and long-drawn-out and indefinite duration. It prevails especially about the Mediterranean basin.

Gentry and Ferenbaugh have recently found a nest of malta fever throughout the older goat-raising sections of Texas. This endemic center embraces an area approximately 300 miles along the Rio Grande extending 90 miles to the north. All the cases of malta fever found have occurred in territory devoted to goat raising, and all the patients there gave a history of drinking unboiled goats' milk or were associated with the goat-raising industry. The *Micrococcus melitensis* was isolated from several of the cases.[2]

Modes of Transmission.—From experimental evidence it would appear that the infection of malta fever may be taken in through wounds, the mucous membranes, or by food and drink. The usual mode of infection is by drinking raw goats' milk. The *Micrococcus melitensis*

[1] "The Origin of the Recent Outbreak of Foot-and-Mouth Disease in the United States," by Mohler and Rosenau, *Cir. 147,* Bureau of Animal Industry, Dept. of Agriculture, 1909.

[2] *J. A. M. A.,* Aug. 26, Sept. 9, Sept. 23, Sept. 30, 1911.

leaves the body in various secretions and excretions. Great numbers of the cocci in pure cultures may appear in the urine. The milk of goats also contains the virus. All the secretions from the body must be regarded as infectious until further knowledge on the subject is at hand. In man the coccus may be isolated from the spleen, lymph glands, bone marrow, and mammary glands. In goats it first disappears from the blood, then the spleen, and, last of all, from the mammary glands.

Goats are susceptible to malta fever and continue to discharge the infection in the milk for a long time. The disease is usually contracted by drinking such infected milk. While this is the common mode of infection, occasional cases doubtless arise through other sources; thus one case which arose in England is supposed to have been conveyed from son to father by using a clinical thermometer in the mouth immediately after its use by the patient. Monkeys may readily be infected either by the inoculation of pure cultures or by feeding them. At least five accidental infections have occurred in bacteriological laboratories, one in Washington. MacFayden lost his life from a laboratory infection with the *Micrococcus melitensis*. This microorganism has, therefore, more than complied with all the requirements of Koch's laws.

There has long been a suspicion that malta fever may be conveyed through the bite of an ectoparasite. In fact, Captain Kennedy was able experimentally to infect a monkey as a result of bites of mosquitoes (*Culex pipiens*) which had fed on patients suffering with malta fever. This probably was an instance of mechanical transference of the infection, corresponding in all respects to a laboratory inoculation with fresh virulent material from a hypodermic syringe. This cannot be a frequent way by which the infection is transmitted in nature, for the specific organisms are found in small numbers in the peripheral blood of malta fever patients. The British Commission found the *Micrococcus melitensis* only four times from a total of 896 mosquitoes dissected.

From the fact that the micrococcus may be successfully introduced either by ingestion, or by inoculation, or through the mucous membranes, it is evident that occasionally cases may receive their infection through a great variety of means, such as insect bites and other wounds, infected food, and the various modes of contact infection. Contact infection, however, probably plays a minor rôle, for there is evidence that the disease is not, as a rule, directly transmitted from the sick to the well. There is little doubt but that the infection can be acquired from the urine secreted by cases of malta fever, and this is probably one way in which the workers in hospitals become infected.

There is also experimental evidence to show that monkeys can be

infected by dry dust artificially contaminated with cultures of the *Micrococcus melitensis*. The path of entrance may be through the nares, throat, respiratory passages, or alimentary canal. Dry dust contaminated with the urine of cases of malta fever has given rise to infection in goats but not in monkeys. The experience gained during the work performed in Malta during 1904 and 1905 has convinced Horrocks that men are more susceptible than monkeys and goats. Shaw's work on ambulatory cases of malta fever among Maltese has also shown that opportunities for the creation of infected dust are plentiful in Malta. Infected dry dust as a mode of transmission cannot, therefore, be discarded, but, as a matter of fact, it probably seldom occurs.

Goats' Milk and Malta Fever.—We are indebted to the six reports of the British Commission for the investigation of Mediterranean fever (1905-1907) for the fact that malta fever is chiefly spread through goats' milk. Before the researches of this commission the common mode of infection was not definitely known.

The usual source of milk in Malta is the goat. The udders, which are abnormally long, often touch the ground and are very liable to be soiled. It was first shown by Zammit in the report of 1905 that goats could be infected by feeding them with the *Micrococcus melitensis*. In the same year Major Horrocks discovered the *Micrococcus melitensis* in the milk of an apparently healthy goat. Further studies showed that one or more healthy goats in every herd were excreting the *Micrococcus melitensis* in their milk and urine, and that about 50 per cent. of the goats reacted positively when examined by serum agglutination tests. All the available evidence points to their food as the main vehicle of infection in goats. The young goats, of course, are infected through their mother's milk. Horrocks and Kennedy consider that 10 per cent. of the goats supplying milk to various parts of Malta excrete the *Micrococcus melitensis* in their milk. The excretion of the specific microorganism may continue steadily for three months without any change occurring in the physical character or chemical composition of the milk and without the animal exhibiting any signs of ill health. On the other hand, the excretion of the *Micrococcus melitensis* in the milk may be intermittent, appearing for a few days and then disappearing for a week or more.

Major Horrocks in *Report No. 5* of the British Commission shows a direct relation between the number of goats in Gibraltar to the number of cases of malta fever. With the reduction in the number of goats in Gibraltar there was also a decrease in the number of cases, so that finally, when the number of goats had decreased to about 200 in 1905, malta fever had practically disappeared.

The story of the steamship *Joshua Nicholson* is instructive in show-

ing the relation between goats' milk and malta fever in man. Sixty-one milch goats, all healthy in appearance and good milkers (many being prize animals), and four billygoats were shipped on board the cargo steamer *Joshua Nicholson* August 19, 1905, at Malta for passage to the United States via Antwerp. Many of the ship's company partook freely of the milk. The officers drank "mixed" milk collected in a large vessel; the members of the crew each obtaining the "whole" milk from one goat in his own separate panikin. Subsequent bacteriological examination resulted in the recovery of the *Micrococcus melitensis* from the milk of several of the goats. Of 23 men on board the steamer who drank the goats' milk on one or more occasions, no evidence whatever is available as to 13, while of the remaining 10, 9 suffered from febrile attacks, 5 of them yielding conclusive evidence of infection with the *Micrococcus melitensis*.

Resistance.—The *Micrococcus melitensis* is readily destroyed by heat. I have shown that 60° C. for 20 minutes is sufficient to destroy this organism in milk and provides at the same time a liberal margin of safety. It is not destroyed at 55° for a short time, but succumbs in one hour; the majority die at 58°; at 60° all are killed. Phenol, 1 per cent., destroys the coccus in 15 minutes. While this micrococcus shows a comparatively feeble resistance against heat and the ordinary germicides, it shows a remarkable resistance to dryness, for it may remain alive in this state for months.

The micrococcus grows well, but slowly, upon artificial culture media. Visible colonies do not appear until about the fifth day. It may be kept alive indefinitely by transplanting to subcultures at frequent intervals. Exceedingly high agglutinating power develops in persons suffering with malta fever—sometimes as high as 1-100,000. In such cases the proagglutinoid zone may appear, that is, the serum may refuse to agglutinate in low dilutions, such as 1-100, but agglutinate actively in higher dilutions, such as 1-1,000.

Prevention.—Our knowledge of the cause and modes of transmission of malta fever makes the prevention of this disease a comparatively simple problem. The infection must first be eliminated in the goats. Until this is done goats' milk should be pasteurized. Patients having the disease should be treated upon the same principles laid down for typhoid fever, in order to prevent the spread of the infection through food fomites and indirect contact. Convalescents should not be released until the micrococcus has disappeared from the urine. General sanitary measures, such as the suppression of flies and mosquitoes, allaying dust, and the promotion of general cleanliness, should not be neglected.

LEPROSY

Leprosy is a contagious disease in the sense that it is probably always communicated directly from the sick to the well, but prolonged and intimate association with a leper ordinarily seems necessary to contract the infection. The degree of the contagiousness varies very much, depending upon conditions not well understood, but it is plain that leprosy shows little tendency to spread in any of the more highly civilized nations practicing personal cleanliness and enjoying the benefits of modern sanitation. Leprosy prevailed in epidemic form in Europe in the middle ages, but the disease has disappeared from central Europe, remaining only upon the fringe of the continent, in Norway, Sweden, Spain, Italy, Greece, Turkey, Russia, and Finland. It is estimated that there are from 5,000 to 6,000 lepers in the Philippine Islands, and there are many cases in China, Japan, and India. The greatest incidence is found among the natives of the Hawaiian Islands, where one in every 30 or 40 have the disease. Leprosy was introduced into the Hawaiian Islands about 1859, and there found conditions particularly favorable for spread. A Government Commission in 1902 [1] took a census of the lepers in the United States and found 278. Of these 145 were born in the United States and 186 probably contracted the disease in the United States. Of the entire number 72 of the cases were isolated and 205 were at large. Brinckerhoff again studied the prevalence of leprosy in the United States in 1909 and found 139 cases. The official figures for 1912 are 146. Although these numbers represent only the cases officially known, it is evident that the disease is not on the increase in our country and that, while it may be contracted here, it is "contagious" with great difficulty. There are three foci of leprosy in the United States: one among the Scandinavians in the region of the Great Lakes, another among the Orientals on the Pacific Coast, and the third on the Gulf Coast, particularly in Louisiana and Florida. According to the official health reports from our Territories and Dependencies, there were in 1912 in Hawaii 696 lepers, in Porto Rico 28, in the Philippine Islands 2,754, in the Canal Zone 7. The number in Guam and Alaska have not been enumerated. It is known, however, that many cases escape tabulation in the official returns.

The cause of leprosy is the *Bacillus lepræ* discovered by Armauer-Hansen in 1874. The bacillus of leprosy resembles the bacillus of tuberculosis in many respects. It stains more readily and decolorizes somewhat more readily than the tubercle bacillus. It differs from the tubercle bacillus in that it grows with difficulty on artificial culture

[1] White, Vaughan, and Rosenau, *Document No. 269*, 57th Congress, 1st Session, 1902.

media and is much less, if at all, pathogenic for the lower animals. Further, lepra bacilli are usually found in dense clusters and in much greater numbers within the cells than is the case with the tubercle bacillus.

The bacillus of leprosy grows with difficulty upon artificial culture media. For years it has evaded all attempts until Clegg [1] in 1909 succeeded in cultivating it in symbiosis with amebæ and *S. cholerœ* upon plain agar and weakly nutrient agar. Clegg based his work upon the belief that the leprosy bacillus derives its nutrition from the products of the tissue cells in which it is mainly to be seen in leprosy lesions. These results have been confirmed by Currie, Brinckerhoff, and Holman in Hawaii and by Duval in New Orleans.

Immunity.—There is no racial immunity to leprosy. The white races suffered severely during the middle ages. Malays and Mongols now appear most liable to the infection, perhaps on account of their mode of life. The disease is remarkable for its prolonged period of incubation and its chronic course. These facts indicate that the body must possess a high degree of resistance to this infection. So far as known, man is the only animal subject to leprosy under natural conditions. Inoculation experiments into lower animals have recently proved successful in the guinea pig (Clegg); the Japanese dancing mouse (Sugai); and the monkey (Duval).

Rat Leprosy.—There is a disease among rats which is a close counterpart of leprosy in man. It occurs naturally in the *Mus norvegicus* and may be transferred by inoculation to the more tractable laboratory white rat. The disease was first observed by Stenfansky in 1903 in Odessa. In the same year Rabinowitsch found the disease among the rats of Berlin, and Dean in 1903 discovered the disease independently in London, and in a later publication (1905) reported success in transferring the infection by artificial inoculation. Since then rat leprosy has been found by Tidswell in the rats of Sydney, Australia, and the England Plague Commission observed the disease among the rats in India. Wherry and McCoy found a number of cases among the rats caught in San Francisco, California.

The proportion of rats infected with leprosy in different localities varies greatly. Thus in Odessa from 4 to 5 per cent., in San Francisco 0.2 per cent., and in Sydney only 0.001 per cent. Currie failed to find leprosy among the rats of Honolulu. The fact that the infection is absent among the rats of Honolulu and present among the rats in Berlin is evidence that it plays no part in the epidemiology of the human disease.

Leprous rats in a late stage of the disease are usually recognized

[1] *The Philippine Jour. of Science*, Vol. IV, No. 77, Apr., 1909. *Public Health Bull. No. 47*, Sept., 1911.

by the presence of patchy alopecia associated with cutaneous and subcutaneous nodules which may or may not be the site of open ulcers. The diagnosis is readily confirmed by microscopic examination of a smear from an ulcer or a nodule, which will show the specific bacillus of the disease in enormous numbers.

Currie[1] has recently shown that rats may infect each other by contact, also that bacilli of rat leprosy may often be demonstrated in the heart's blood of infected rats. Currie had no difficulty in demonstrating the presence of acid-fast bacilli in mites contained on the bodies of rats when the latter's heart's blood contained the microorganisms. The fact that these mites so frequently contain the bacilli naturally leads to the suspicion that they may be one of the means of transmitting the disease from rat to rat, but up to the present time no positive evidence has been adduced that such is the case.

In this leprosy-like disease of rats we have an infection which closely resembles leprosy in man. The fact that the infection may readily be propagated in a laboratory animal permits of its investigation, and it is assumed that the further studies now being made upon rat leprosy will throw much light upon the modes of transmission and control of the human disease.

Modes of Transmission.—The leprosy bacillus is found in all the lesions of the disease—the nodules on the skin and mucous membranes, in the spleen, liver, and testicles—in fact, in all the internal organs. In the anesthetic type the bacilli are found in the cells of the spinal cord and brain and also in the peripheral nerves. Leprosy bacilli may also be found in the circulating blood during the acute eruptive stage. Frequently they are in the endothelial or in the white blood cells. The leprosy bacillus leaves the body from any of the lesions that are broken down. From the degenerated nodules of the skin or mucous membranes they are discharged in enormous numbers. If we may depend upon microchemical evidence, it appears that most of these bacilli are probably dead. Leprosy bacilli also occasionally appear in the feces and urine. They do not occur in the expectoration from the lungs.

There is some doubt as to just how the leprosy bacillus enters the body, whether through wounds of the skin or through the mucous membrane of the nose and throat or through the digestive tract, or possibly during coitus.

It may be definitely stated that leprosy is not due to the eating of any particular food, such as fish. This theory has been stoutly maintained by Jonathan Hutchinson. There is no satisfactory evidence in support of the fish theory and many facts against it. One thing is plain, and that is, leprosy is not contracted from any of the lower

[1] U. S. Pub. Health and Mar. Hosp. Ser., *Pub. Health Bull. No. 50.* Oct., 1911.

animals, but is an infection which in all cases passes rather directly from man to man.

The suspicion that parasitic insects may play some rôle in the transmission of leprosy has existed for some time. The evidence is reviewed by Nuttal,[1] who says: "It appears that Linnæus and Rolander considered that *Chlorops* (*musca*) *lepræ* was able to cause leprosy by its bite." Blanchard and Corrodor tell of flies in connection with leprosy. Flies frequently gather in great numbers on the leprous ulcers and then visit and bite other persons. An observation by Boeck of the presence of *Sarcoptes scabei* in a case of cutaneous leprosy led Joly to conclude that these parasites might at times serve as carriers of the infection. Pediculi are usually present among the poor classes in Algeria, which furnish the greater number of lepers. Sommer of Buenos Aires expresses the belief that mosquitoes act as active agents in the spread of leprosy in warm countries. Carrasquillo of Bogota found the bacillus of Hansen in the intestinal contents of flies. W. C. Goodhue and his assistant, Father Joseph, working at the leper settlement at Moloki, found lepra bacilli in the intestinal contents of a female *Culex pungens.* Later they found similar organisms in the common bedbug. The British Leprosy Commission investigated the possible rôle played by insects with entirely negative results. Wherry studied the occurrence of lepra-like bacilli in certain flies and their larva. He found that the fly *Chlorops vomitoria* took up enormous numbers of lepra bacilli from the carcass of a leper rat and deposited them with their feces, but the bacilli apparently do not multiply in the flies, as the latter are clear of bacilli in less than 48 hours. Larvæ of *Chlorops vomitoria* hatched out in the carcass of a leper rat become heavily infested with lepra bacilli. If such larvæ are removed and fed on uninfected meat they soon rid themselves of most of the lepra bacilli. A fly, *Musca domestica,* caught on the face of a human leper was found to be infested with lepra-like bacilli. Most of the evidence bearing on the possible rôle of insects in the transmission of leprosy may be classified as purely presumptive evidence based upon analogy, or as evidence based simply upon the finding of acid-fast bacilli in certain insects. It must be plain that the simple taking up of parasites by an insect does not necessarily imply that the insect plays a rôle in its transmission from one host to another. Further, all acid-fast bacilli are not leprosy bacilli. It cannot be denied that leprosy may be one of the insect-borne diseases; the final verdict will depend upon further studies.

A great majority of lepers at some time in the disease have lepra bacilli in their nasal secretions. The importance of the nose in leprosy was brought into prominence at the First International Leper Confer-

[1] *Johns Hopkins Hospital Reports.* 1900, VIII, p. 1.

ence in 1897 by the work of Sticker, who made sweeping statements concerning the nose as the site of the primary lesion and the danger of nasal secretions in transmitting the disease. Jeanselme and Laurans (1895), Gerber (1901), Werner (1902), Sheroux (1903), and others have shown the frequency with which the bacilli of leprosy appear in the nasal secretions and the importance of the nose as a site of leprous lesions. Sticker cites a five-year-old child of leprous parents seen by him in India with an ulcer on the right side of the nasal septum which contained lepra bacilli and was the only lesion of the disease present in the case. Plumert (1903) mentions the finding of lepra bacilli in the nasal secretions of persons in intimate family contact with advanced cases of leprosy. The individuals in question showed no other evidence of the disease. Falkao observed epistaxis associated with small ulcers on the nasal septum of descendants of lepers, and lepra bacilli were found in the crusts from these ulcers. The results of Sticker, Plumert, and Falkao would indicate that in the early stages of the disease the nose is frequently the site of a lesion discharging lepra bacilli. Brinckerhoff and Moore, however, who made a careful study of this question in Honolulu, point out that most of the studies upon the importance of the nose in leprosy have been made upon relatively advanced cases of the disease. They found the nose frequently the seat of infection when the disease is well developed, but practically never as a primary or incipient lesion. If the nose were the usual seat of the primary lesion in leprosy, it would indicate that the infection is carried there upon the finger.

Hollmann studied 500 persons in the Hawaiian Islands suffering with a recognizable form of leprosy for periods varying from three months to twenty-five years, and found 410 with lesions of the nasal mucous membrane and only 90 in which such lesions were absent.

It is sufficient for practical prevention to know that leprosy is spread mainly by direct contact and perhaps occasionally by indirect contact with persons suffering with the disease. Leprosy is most prevalent under conditions of personal and domestic uncleanliness and overcrowding, especially where there is close and protracted association between the leprous and the non-leprous. There is no evidence that leprosy is hereditary. The occurrence of several cases in a single family is doubtless due to "contact." The danger of infection from leprous persons is, of course, greater when there is a discharge from the lesions of the skin and mucous membranes.

Prevention.—The prevention of leprosy depends almost entirely upon isolation, care of the infected discharges, personal cleanliness, and sanitary surroundings. That the disease is transmitted with difficulty is shown by the fact that doctors, nurses, sisters of charity, ward tenders, and others directly exposed in leprosaria seldom become

infected. Notable exceptions have been Father Damien in Honolulu and Father Bogliolo in New Orleans. Evidently close, prolonged and intimate contact is ordinarily necessary to contract the infection. For many years a case of leprosy was cared for as a patient in a hospital with which I was associated. He had his own dishes, towel, soap, etc., otherwise he mingled freely with the patients and others, without spreading the disease.

For the control of leprosy the most important administrative measure is to segregate the lepers in settlements or asylums. Compulsory notification of every case of leprosy should be enforced, if for no other reason than to keep track of the disease and to know whether it is on the increase. Segregation of lepers is the most important single preventive measure. The leprosaria should be inviting and should contain all modern improvements for the care and treatment of the disease. Leprosy is by no means invariably fatal. In the United States, where there are only a few hundred lepers, the Government should establish a national leprosarium conducted upon the principles of a modern sanitarium for tuberculosis. To require each state to provide suitable accommodations to segregate its few lepers is economically wasteful. It is claimed that the decrease in leprosy in Europe since the middle ages has been due in large part to the segregation of the lepers in leprosaria, which at one time numbered 20,000. On the other hand, the value of segregation in countries where leprosy is very prevalent is disputed. As a rule, only the advanced cases are detected and isolated. Segregation in the Hawaiian Islands has so far had no effect upon the prevalence of the disease. There are factors in the control of leprosy not yet understood.

There can be little objection in a country such as ours, where leprosy shows slight tendency to spread, to give a clean leper his freedom. There is no more danger from a leprosy patient of clean personal habits, who exercises care concerning the discharges from the lesions, than there is from a discharging case of tuberculosis of the glands of the neck.

The national quarantine regulations forbid the landing of an alien leper. The law requires that such person be deported on the same vessel that brought him. A citizen of the United States having leprosy cannot be debarred. Such individuals are admitted and then come under the health laws of the state or port of entry.

Specific Prevention.—There is no specific prevention or cure for leprosy. Nastin is a substance proposed by Deycke and consists of a neutral fat obtained from a streptothrix. The reports from its use are not particularly encouraging. Rost, of Rangoon, Burmah, uses a substance which he calls "leprolin," a precipitate from leprous tubercles. Tuberculin in somewhat large doses injected subcutaneously

into leprous patients produces both a general and local reaction, but the repeated injections do not materially influence the disease, although such treatment seems to cause a local improvement or softening of the leprous tubercles. Heiser in Manila reports favorable results from the application of X-rays. Dyer in Louisiana has obtained good results from good food, fresh air, cleanliness, and the general principles applicable to the modern treatment and prevention of tuberculosis.

MENTAL DISEASES

THE PREVENTION OF MENTAL DISEASES

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Although, in the prevention of insanity, we have to deal with problems more complex than those which have been considered in the prevention of the infectious diseases, some mental diseases are known to depend upon causes as definite as the infection of the body with pathogenic bacteria. We know, for instance, that if a patient with one of the alcoholic psychoses had not been addicted to the use of alcohol he would not have acquired this mental disease, whatever other bodily or mental infirmity he might become afflicted with, for recent methods of studying mental diseases have made it possible to recognize certain groups of symptoms which can be produced by alcohol and by that cause alone. There are clinical symptoms and laboratory findings which enable us to learn, with small chance of error, that a patient is suffering from general paresis. We know that such a patient owes his mental disease to syphilis, and that for him the prevention of insanity would have consisted in the prevention of syphilis.

Not all types of mental diseases, however, have causes so well understood as these. There are many in which the pathology is unknown and in which the symptoms are so variable that at present we are obliged to place them in provisional groups from which we may be able to rescue them later, perhaps, when present diagnostic difficulties have been overcome or when new light has been thrown upon their nature. It seems desirable, in discussing the preventable causes of insanity, to consider first some of the factors which we know are capable of producing mental disease, either directly or indirectly, at the same time referring to possible means for their control, and then to consider some other causes which we have excellent reasons for believ-

ing influence the prevalence of insanity, but which operate in a manner which cannot be shown so conclusively.

It is essential to state at the outset that a number of different diseases are included in what we term "insanity." It would be quite permissible to speak of the various mental diseases as "insanities," so greatly do they vary in their symptoms, course, and etiology. Just as the popular term "heart disease" properly includes congenital malformations, changes associated with acute infectious diseases, reactions to toxic substances, disturbances of the nervous mechanism, and the effects of disease of remote organs, so "insanity" includes diseases dependent upon congenital mental deficiency or developmental defects, the exhaustion accompanying acute or chronic disease, the introduction of toxic substances into the body or their elaboration within it, organic changes in the brain, and abnormal psychic reactions.

INFECTIOUS DISEASES WHICH CAUSE INSANITY

It seems desirable to consider infectious diseases as a cause of mental disease first, not because they are responsible for a larger proportion of cases than other preventable causes, but on account of their closer relation to that with which we are familiar in the realm of preventive medicine.

Typhoid fever may give rise to permanent or transitory mental impairment. The prevention of insanity in this instance consists, of course, in the prevention of typhoid fever. When the evils resulting from the needless prevalence of that disease are counted up, the cases of mental disease caused by it must be included.

Other infectious diseases, notably *influenza, scarlet fever, malarial fever, erysipelas,* and *septicemia* (particularly from uterine infection), furnish a considerable number of cases of mental disease, chiefly in the infective-exhaustive group, in which exhaustion, elevated temperature, and poisoning of the nervous centers by bacterial toxins are the immediate causes of mental changes. About three per cent. of all first admissions to hospitals for the insane [1] belong in this group. It is impossible to estimate the proportion of cases in which infectious disease is the *only* cause in other types of mental disease, for an acute infection may "liberate" an attack in a patient subject to a psychosis in which recurrences are common, and this cause may combine with others, alcohol, for instance, in the production of a psychosis in which the infectious disease plays a secondary part.

[1] This percentage and others following are based upon recent statistical studies of admissions to the state hospitals of New York and Massachusetts. About one-fourth of all the insane under treatment in the United States are patients in the public institutions of these two states, and statements based upon the statistical studies in question are fairly applicable to the United States as a whole.

Preventive measures in such types of insanity must consist chiefly in the general work of limiting the prevalence of the infectious diseases, but much can be done by improved methods of treating febrile conditions. The full significance of delirium and its pathology must be appreciated and hydrotherapy employed more generally and more carefully if we are to lessen the number of patients with infective-exhaustive psychoses. The indiscriminate use of sedatives or hypnotic drugs in deliria sometimes results in an aftermath of mental disease.

Syphilis deserves separate consideration as a preventable cause of mental disease, for it is the essential cause of general paresis, a disease responsible for about 13 per cent. of all first admissions to hospitals for the insane in this country, and for nearly one-fifth of all male admissions. In admissions from American cities more than 22 per cent. of male patients are suffering from general paresis. More deaths resulted in New York State from general paresis in 1911 than from smallpox in the whole registration area of the United States since 1908. Half as many deaths are known to occur every year from general paresis as from typhoid fever. It is believed that a considerable number of deaths from general paresis, when occurring outside of institutions, are reported as "softening of the brain" or by some other indefinite term, and the prevalence of general paresis is, therefore, far greater than mortality statistics would indicate.

This disease runs a uniformly fatal course, the average duration of which is from two to five years. It attacks people who have, to all appearances, recovered from syphilis, and most frequently in the fourth decade of life, when their usefulness to the community and to their families is greatest. It is the grimmest specter which follows youthful indiscretions and "sowing wild oats." Of course, the prevention of general paresis depends wholly upon the prevention of syphilis, a well-defined field of effort in preventive medicine, but it seems that impetus would be lent the movement for venereal prophylaxis if the appalling prevalence of this result of syphilis were more widely known. It is a rather surprising fact that many of those actively engaged in the campaign against venereal disease are quite unaware of the prevalence of general paresis or that it depends upon previous infection with syphilis. It is a fact that general paresis is a much more frequent manifestation of syphilis than locomotor ataxia.[1]

[1] It is very desirable to know what proportion of cases of syphilis result in general paresis, but, until recently, no satisfactory studies had been undertaken to determine this, and, on account of the long interval between infection with syphilis and the development of symptoms of general paresis, it seemed impossible to find a group of population in which such studies could be made. A short time ago, however, Mattauschek and Pilcz made public (*Berliner. klinische Wochenschrift*, Feb. 19, 1912) the results of a careful examination of the histories of 4,134 officers of the Austrian Army who had contracted syphilis during the period 1880-1890. They ascertained that 4.67 per cent. of these officers developed general paresis.

A small number of cases of other types of mental disease are also directly the outcome of syphilis. Mental deterioration is associated with gummata of the brain and mental changes accompany local meningitis due to syphilis. Syphilis is also responsible for a certain proportion of cases of congenital mental defect upon which insanity may become engrafted later, and it is often syphilis which first attacks the integrity of the arterial wall, thus laying the train destined to result, years later, in arteriosclerosis and mental disease dependent upon it.

Tuberculosis is a cause of mental disease much less frequently than has been supposed. It is exceedingly doubtful if, as has been asserted, tuberculosis ever results in a special clinical form of mental disease, but the exhaustion of a chronic, wasting illness and the action of the tubercular toxin upon the nervous centers are probably immediate causes of mental changes. Although there are no especial measures of prevention of mental disease dependent upon tuberculosis, the fact that this is one of its possible effects might well be added to the information disseminated regarding tuberculosis, for not the least of the benefits from curing incipient cases or preventing the spread of tuberculosis is that the prevalence of insanity is thereby even slightly lessened.

Pellagra.—It is hardly justifiable, perhaps, to speak of pellagra as a preventable disease when the ground is just being cleared for a satisfactory search for its cause, which at present is assumed to be spoiled corn (see page 577). With the practical application of means for the control of this disease, a certain number of cases of insanity will be prevented in the localities where pellagra prevails.

ACUTE AND CHRONIC POISONINGS WHICH CAUSE INSANITY

Alcohol.—It is a strange commentary upon human frailty that all the poisons which assail man through accident and the dangerous trades in which he must engage, and all the poisons which are elaborated within his system, as in nephritis, diabetes, thyroidism, and acromegaly, are together responsible for but a small fraction of the number of cases of mental disease due to his deliberate ingestion of one poisonous substance—alcohol.

It is likely that alcohol, as a predisposing or as an immediate cause, is responsible for more than a third of all admissions to our hospitals for the insane. When, however, we consider alcohol as a cause in diseases in which other etiological factors enter, we are upon ground where statements must be made with caution and with many qualifications. Thus a man with a considerable degree of congenital mental defect is induced by some companions to take a few drinks

of whiskey, and he thereupon develops an episode of excitement which lasts several months. Alcohol is not the most prominent feature in such cases, perhaps, and yet if it is withheld such persons might never develop acute mental symptoms. In considering alcohol as a cause of mental disease it seems best to confine ourselves at first to those diseases which, from their symptom-complexes, we have come to recognize as the alcoholic psychoses. In these disorders, acute alcoholic hallucinosis, chronic alcoholic insanity, and Korsakow's disease, to diagnose the disease is to know the cause. About 12 per cent. of all first admissions are for these psychoses.

They are met in men about three times as frequently as in women, and, as in the case of general paresis, more frequently in admissions from cities than from the country, although there is by no means as much disparity. These alcoholic psychoses are the direct, unmistakable results of intemperance, acting in many cases upon psychopathic individuals, but it is believed that in less direct ways alcohol is responsible for nearly as large a share of admissions to hospitals for the insane. In the year ending September 30, 1909, alcohol was assigned as an etiological factor in 31.4 per cent. of the men admitted to the New York State hospitals, and in 9.6 per cent. of the women. As a habit disorder, but not a definite etiological factor, intemperance was reported in 14.3 per cent. of cases among male admissions and 6.1 per cent. among female admissions. So 45.7 per cent. of all the men admitted and 15.7 per cent. of all the women admitted were addicted to the excessive use of alcohol. This is a prevalence of intemperance enormously greater than in the general population, but it must be remembered that not a few patients admitted to institutions for the insane had become intemperate as a *result* of mental disease, and a great number, including those with alcoholic psychoses, as a result of constitutional mental inferiority. The idea is spreading among psychiatrists that, *in a world of drinkers, the alcoholic is an abnormal type.* This fact does not in any way lessen the importance of alcohol as a cause of mental disease, but it shows the great necessity of throwing especial safeguards about unstable persons in whom intemperance may lead to such disastrous results.

There is hardly a mental disease which is not influenced unfavorably by alcoholic habits. They lend a tremendous impetus to the retrogressive changes in senility, and, as has been said, the acquisition of alcoholism by defectives often results in acute mental symptoms when none need have occurred. Statistics collected independently by several investigators show that the parents of nearly 50 per cent. of defective children were alcoholics. It is held by many psychiatrists that no other single cause of imbecility and idiocy except mental defectiveness in the parent can compare with alcoholism in the parents, intemperance of

mothers during pregnancy being thought to be particularly likely to result in mental defect in the offspring.[1]

The prevention of mental diseases due to alcohol, like the prevention of those due to syphilis, is only part of the general movement against these enemies of the race. Excluding poverty and crime, there is probably no more disastrous result of alcoholism than the continual procession of unfortunates who are entering hospitals for the insane because of intemperance, and it is certain that no other fatal termination of syphilis is so frequent as general paresis.

Other Exogenous Poisons.—Morphinism and other drug addictions are responsible for less than one per cent. of first admissions to hospitals for the insane in this country. Fewer such patients are admitted in New York and Massachusetts now than were a few years ago. This gratifying fact is due, in part at least, to stricter enforcement of the laws regulating the sale of narcotics and particularly to the pure food laws which have rendered it a little more difficult to dispense habit-forming drugs in patent medicines. It is well within our power to eliminate this cause of mental disease by wise legislation and its rigid enforcement.

A very small proportion of admissions is caused by occupational poisonings, such as lead and carbon monoxid. This small proportion can be still further reduced by increasing attention to measures safeguarding workmen in dangerous trades.

Endogenous Poisons.—Those poisons originating within the system which produce mental disease are for the most part the result of other diseases, and these diseases—nephritis, heart diseases, and diabetes—are, unfortunately, beyond the reach of preventive medicine. When the infectious diseases have come under our control, and when means have been devised to reduce accidents to a minimum, the preëminence of these diseases as causes of death will only be accentuated.

In the case of psychoses dependent upon diseases of the thyroid gland, early treatment of the primary disease is a hopeful means of prevention. It is curious that vaccines and sera should be furnished free by the state while any person can remain mentally defective from cretinism, because prolonged treatment is too expensive. The state has such an enormous number of the insane and mentally defective

[1] It should be said that recent studies by the Francis Galton Laboratory of Eugenics, London, point to directly opposite conclusions. The weight of evidence, however, is in favor of the relation between alcoholism and mental defect indicated above. Bezzola and Hartmann state that examinations of the birth-dates of idiots and imbeciles in Switzerland show that conception recurred in a large proportion of cases at seasons of the year when the celebration of certain festivals were accompanied by much intoxication. It is said that this is popularly recognized and that such children are known in certain districts as *rausckinder* (''jag-children''). On the other hand, the birth-dates of defective children in certain fishing villages in Northern Europe where there is much periodic intoxication have been carefully studied and no such relation discovered.

for whom permanent care must be provided that it would be a matter of sound policy to seek out such cases and then provide the best care and treatment absolutely without cost.

HEAD INJURIES AND INSANITY

A considerable number of the cases admitted to every large public institution present a history of head injuries, usually with fractures of the skull. There are certain symptom-complexes especially frequent in such cases, and so it can be said that injury of the brain is a specific cause of mental disease. Street accidents, railroad accidents, and unprotected machinery are by far the most frequent causes of head injuries in civil life. It is quite justifiable to consider this cause of insanity as preventable, for one has only to read the recent literature on safeguarding workmen in factories and protecting railway employees to see that this is a field in which much may be done. Street accidents have been practically eliminated in some cities by efficient police regulation of traffic. In a consideration of the prevention of insanity this phase is not sufficiently important to receive much space, and yet it seems desirable to mention it.

HEREDITY AND INSANITY

It is probably safe to say that none of the causes of insanity which have been considered, unless it be alcohol, is as important a factor as heredity, and heredity enters largely into the production of alcoholism, or, at least, into the production of the mental type which succumbs to alcohol. Opinion as to the influence of heredity upon the development of mental disease has undergone much change in recent years, as it has in reference to other diseases, but it can be said that studies which have led to heredity being considered of secondary importance in some conditions in which it was thought to be paramount have been offset by studies in other directions which have disclosed heredity as a factor of the greatest importance. Studies are actively under way to determine the relation of Mendel's laws to heredity in insanity. In large series of admissions for all forms of mental disease in this country and in Europe it has been found that, in the cases in which a satisfactory history was obtainable, about 50 per cent. of the patients had an insane heredity. There are no statistics available in this country to show the proportion of normal people with an insane heredity, but Kraepelin quotes Jost as giving the percentage as 3 and Nacke as giving it as 7.5. There are some mental diseases in which the percentage of insane heredity is only slightly more than this, while in manic-depressive insanity the percentage has been found in a large series of cases to be as high as 70, many of the insane ancestors having other forms of mental disease.

This shows very interestingly the unity of some of the factors which underlie mental diseases of different types.

Without discussing the influence of heredity further, some possible means of prevention may be considered. This is the domain of eugenics. The means suggested all have for their object either the permanent sequestration of the insane and mentally defective or the prevention of offspring by control of marriage or by sterilization. It has been proposed by some that it should be required by law that no woman in the child-bearing period who secures admission to a hospital for the insane for a psychosis in which heredity is known to play a prominent part should be discharged, even if recovered, until the menopause has been reached, and it is asserted that the welfare of the race justifies such a procedure. It cannot be denied that such a course might in time effect some reduction in the prevalence of mental diseases. It would seem that society can find justification for adopting some such measures to protect itself in those cases in which every period of parole or discharge from a hospital is followed by a pregnancy, but the ethical considerations involved are so complex that it is sufficient here merely to state this proposal. It has been proposed to sterilize by vasectomy or other means all patients when they are about to be discharged from a hospital for the insane, if their mental disease was of a type in which heredity is prominent. Here again there are complex ethical questions to be considered, but, as an alternative, it may be suggested that all such patients should at least *be offered* the opportunity of providing against such an occurrence. It is believed that, if the matter were tactfully and earnestly presented, the freedom from danger pointed out, or even a small bounty paid by the state in suitable cases, there would be a considerable number of acceptances.

Prohibiting the marriage of those in whom insane heredity within a close degree of relationship exists would depend for its effectiveness upon the provisions for obtaining such information and any such measure would be practically without value unless it were required in a number of states. Education as to the dangers of heredity and appeal to social conscience, by physicians, teachers, and clergymen, would undoubtedly deter a few from marriage, but it is the experience of physicians generally that such advice is rarely heeded. Before the matter can be presented in a "campaign of education" there is need for far better information than we possess at present. Facts should be most carefully sifted and statistical studies of broad scope undertaken under the auspices of the government or some national society before information regarding heredity and insanity is prepared for wide dissemination.

PSYCHICAL CAUSES

During the last few years the psychical causes of insanity have been recognized as of great importance, and types of mental disease which were thought to be almost wholly dependent upon the constitutional make-up of individuals have been shown by Freud, Jung, Meyer, Hoch, and others to be dependent very largely upon errors of education, unsuitable environment, the acquisition of injurious habits of thought, and the suppression of painful experiences, usually in the sexual field, which later in life form the basis for psychoses. The outlook for the prevention of insanity is very hopeful in some of these cases *in the individual.*

Emphasis must be placed upon the important fact that the foundations of aberrations in which sexual trends are prominent are laid at an extremely early age. It is only very recently that it has been shown that experiences in childhood and infancy exercise a controlling influence upon the sexual life of later years. The practical application of this is not that children are to be brought up in seclusion, but that there is no higher duty of parents than to establish such relations with their children that sex difficulties can be discussed and straightened out before they give rise to permanent moods or trends. Hypocrisy and false shame are not natural attributes of the child, and when we create them we raise a barrier behind which much damage may take place without our knowledge. In childhood and in adolescence there must be established the closest bonds of sympathy and understanding between parent and child. Wholesome, frank communication and sensible consultations at that time regarding sex quandaries may save a child or young adult from disaster later on. In the aberrations which come with adolescence one can usually recognize the results of early mismanagement in these matters, and the urgent need for sound advice and correct guidance needs no elaboration.

In dispensaries for "border-line" cases some hidden sexual trends or other factors in psychogenesis may be discovered, and preventive measures prove successful. Nothing could aid more in the discovery of such factors than general appreciation by physicians that they may exist and may result in insanity. If there were such general recognition of the part such factors play many persons not insane would be referred to the psychiatrists and more psychiatric clinics would be established.

ECONOMIC FACTORS

Unemployment, overwork, congestion of population, child labor, and the hundred economic causes which increase the stress of living

for the poor are factors in the production of insanity which often seem to outweigh all others. Weaknesses in constitutional make-up—defects in the armor of personality—are disclosed under the stress of such conditions which would have remained undiscovered under happier circumstances. All that can be said of the prevention of such causes is that everything which makes for the betterment of those upon whom the stress of living falls heaviest will save many from mental disease. For the individual careful training, encouragement, wise counsel, and a little financial assistance in times of especial need are helpful measures. If the operation of these powerful causes cannot be prevented, those who are most likely to be harmed would, perhaps, be shielded a little if the dangers which they face were more generally known.

IMMIGRATION

No consideration of the preventable causes of insanity in this country would be complete without reference to this important element in our national life. It is a question peculiar to the United States. Since 1820 more than 28,000,000 immigrants have come to this country. This vast migration has no parallel in history. In some states the increment to the population from immigration every year exceeds that from births. Under such conditions movements such as those directed against alcohol, heredity, or the economic causes of insanity are feeble compared with a thorough sifting of applicants for admission while they are still at our threshold. We have the absolutely unquestioned right to require any reasonable tests which can be proposed, and yet the present immigration law results in the mental examination of only *one in every thousand* of the million immigrants who seek admission each year. There is no provision whatever requiring immigrants to present certificates from responsible authorities at home, testifying to their freedom from mental disease. These crowds of immigrants, 30 per cent. of the adults illiterate and less than 20 per cent. with any trade, are, practically without mental examination or selection, projected into our most congested centers of population, to bear, during their first year in America, as severe stress as any group of population can be called upon to endure. One result is that they flood our hospitals for the insane. Hundreds have to be returned during the first year for mental disease due to causes which existed before their arrival. In the succeeding years the proportion rises and in the next generation and the one succeeding it we shall reap the harvest for which our present policy is sowing the seed. It can be earnestly asserted, after long study of this question, that no measures for the prevention of insanity which have yet been suggested can prove so efficacious as artificial selection of accretions to our population, on the vast scale which an

adequate mental examination of immigrants would permit. This is a measure of practical eugenics which can be applied as successfully now as in a generation. As Professor R. DeC. Ward has said, "it is merely a question whether we or foreign steamship agents shall select the parents of future generations of Americans." The provisions of the federal immigration law which deal with the exclusion of insane immigrants are in need of thorough and immediate revision, and the enforcement of the law should receive the attention which its importance deserves.

We have been far too careless of the welfare of recently landed immigrants. There seems to be a general impression that, however unsanitary their surroundings or however heavy may be the burdens placed upon them, immigrants are, in some way, fitted for such hardships, either by nature or through previous experiences in their homes. Of course, this assumption is wholly without justification, and it is time that the social, economic, physical, and moral welfare of these newcomers be given the earnest attention of the federal and state governments and of societies and individuals. By so doing something may be done to lessen the terrible prevalence of mental disease in this large group of our population.

THE AGENCIES AVAILABLE FOR THE APPLICATION OF PREVENTIVE MEASURES

It is possible to outline only very briefly the agencies which can be utilized in the application of preventive measures.

Hospitals for the Insane.—A very large proportion of the insane persons in any state will be found under treatment in public institutions. This is not the case with other diseases, sufferers from which are widely scattered, in their homes, at work, and in hospitals. This fact makes the hospital for the insane seem the logical place from which preventive measures should emanate.

Every large hospital for the insane, unless entirely inaccessible, should maintain a dispensary. To that dispensary, if it is skillfully conducted with its broadest aims constantly in mind, will come incipient cases, "border-land" cases, those who have had previous attacks of mental disease, and relatives seeking advice. Such dispensaries—and several have already been instituted—afford rich opportunities for the practical application of preventive measures and for the dissemination of information. Members of the hospital staffs should also engage in field work in the districts from which their hospital receives its patients. Talks on the preventable causes of mental disease, the advantages of earlier treatment, and the necessity of considering insanity as a disease and not a crime can be given by such medical field workers in

schools and churches and before clubs and societies. Such talks should be supplemented by illustrated descriptions of modern methods of caring for the insane and promoting their happiness and comfort. It is usual for citizens to have a local pride in their hospital as a public institution, and this will often insure interest. Every such lecturer will be quite sure to have the relatives or friends of some of his patients among his hearers. Such field work by physicians should be supplemented by that of well-trained social service workers permanently attached to the institution.

The hospital should also be the center for instruction in clinical psychiatry in the community. The great wealth of clinical material in a large hospital for insane should be utilized to the fullest extent if a medical school is near enough, but it is believed that better knowledge of mental diseases in this country will be brought about much more effectively by developing the opportunities of the general practitioner for receiving instruction than by increasing very greatly the time devoted to psychiatry in medical schools. The medical student is often overburdened, and he has much difficulty in deciding upon the relative value of the matters presented to him. In competition with other branches of medicine psychiatry is very apt to fare badly, for it is likely to be regarded as a specialty of slight value or interest to one who is about to engage in general practice. With the practitioner it is different, for he is a dull man who does not learn early in his career that mental diseases are frequently met with and are very important in many of their relations. It is a fact that there is no opportunity in the United States for a graduate in medicine to obtain post-graduate instruction in psychiatry unless he is a member of the staff of an institution for the insane or a medical officer of one of the government medical corps.

The hospital for the insane has many opportunities for instructing general practitioners. Frequent medical meetings at the hospitals, in which clinical talks should have chief place, correspondence with physicians who sign commitment papers regarding interesting features or the course of their cases, invitations to necropsies (which in most small communities will be gladly accepted), and consultations when patients are about to be discharged, at which suggestions for after-care can be made, are all means of interesting physicians in mental diseases and their prevention. All these new tasks, which are certain to be assigned to our hospitals for the insane within a few years, will necessitate additional medical officers, and they make it more necessary than ever that clinical and laboratory work in these institutions should be upon a high plane. This means increased appropriations, but it is doubtful if a state can utilize its funds for a better purpose than in fostering the work of prevention in mental diseases. Placed upon a purely eco-

nomic basis, such work is immensely profitable. It has been estimated that prevention of the admission of a single patient each year would yield a return to a state larger than the pay and expenses of two social service field-workers for a year.

The hospital has also excellent opportunities for disseminating information among the laity regarding the cause and prevention of insanity. Leaflets, personal talks, and general literature regarding these subjects will not fail to interest those who have come to the hospital to visit a near relative.

Central Boards of Control.—In most states the administration of hospitals for the insane is in some measure under the control of a central board. Such bodies can do much in the prevention of insanity. In many states they can require such activities on the part of the hospitals as have been outlined, and in others they can exert powerful moral influence in having them undertaken. They can conduct statistical studies as to the preventable causes of insanity, and secure wide distribution of the material collected. They can suggest and urge legislation for the early treatment of the insane and for the adoption of specific preventive measures. They can, particularly by coöperation with similar authorities in other states, secure some reforms in federal legislation regarding the exclusion of insane and mentally defective immigrants, the urgent need for which has been pointed out.

National and Local Societies for Mental Hygiene.—There is a very clearly defined field of effort for national and local societies in the work of prevention of mental diseases. As has been indicated, the care of the insane is, far more than that of any other class of the sick, in official hands. There is besides a great deal in the methods of commitment and provisions for care, which is regarded wholly as an official matter. For this reason there is decided need of agencies which can bridge the gap between the home and usual environment of the patient and the public institution which is charged with his care. A certain part of the social service work which has so useful a place in the care of the insane, particularly in the period following discharge from institutions, should be done by workers under the direction of institutional authorities, but there is also a very great deal which can be done better by societies coöperating with institutional authorities but not officially connected with them. In New York State the "Committee on Mental Hygiene" of the State Charities' Aid Association has a local committee in each hospital district. Although after-care and efforts to improve the kind of care afforded the insane in that critical period while commitment is pending constitute the chief work of such committees, there is often opportunity for effective work in prevention. In Connecticut, Illinois, and Massachusetts there are state societies of mental hygiene doing most useful work.

There is a National Committee for Mental Hygiene, coördinating and, in a measure, directing these local activities. This committee has commenced studies into existing provisions for the care of the insane in all the states, methods of commitment and care, the influence of preventable causes, etc. With a carefully prepared plan of work, accurate information is to be obtained upon these matters, and, as fast as the facts in the possession of the committee justify it, active work is to be undertaken for amelioration or prevention. It is believed that a great deal can be done, especially in the direction of standardizing work for the care of the insane and the prevention of insanity, and in coördinating the efforts of the hospitals, state boards of control, and some of those organizations which sometimes, perhaps unawares, are attacking preventable causes of insanity from different angles. Such an organization as the National Committee for Mental Hygiene can stimulate interest on the part of the state and local authorities charged with the care of the insane, and it can sustain interest when it might otherwise flag. Standards established in a state where advanced ideas prevail can be made known in states where there is indifference or lack of progress. A central "clearing house" for the collection and distribution of accurate information regarding the care of the insane and the prevention of insanity can be provided. Earlier treatment and the transfer of care pending commitment from the policeman to the doctor—the most urgent needs of the insane—can be secured by this organization, and the lamentable failure to provide instruction in mental diseases in the medical schools can be shown, and the means suggested for remedying the defect. It is a fact that the number of beds in the institutions for the insane in this country is greater than the number of beds in all the general hospitals of the United States. The insane are, therefore, the most numerous class of the sick receiving public care. As such, they demand a large share of the interest of every practitioner. Progress in every branch of preventive medicine depends most upon the efforts of physicians, and in this particular field there is need of much wider interest on the part of the medical profession than exists to-day.

Education.—Under the direction of state boards of control and encouraged by national and state societies for mental hygiene, much can be done toward placing the education of defective children upon a better basis. These children are now chiefly interesting to school authorities, for they constitute a special class and should receive separate instruction, both for their own good and the good of normal children, whose progress is retarded on account of the excessive amount of time teachers must give defective children. They should have a far greater interest for the state than this, for every such child is a possible patient in a hospital for the insane or in a colony for the mentally defective. Every effort to prevent this outcome is justified, and it would seem

desirable for the state to provide very liberally for the study of these children and for their education. Of even more importance, perhaps, is the permanent segregation of most of them.

It has been estimated that in a state which adopts a high standard of caring for the insane about one-fifth of the annual income of the state will be required. Whether insanity is increasing or not, the number of the insane under treatment has, up to this time, increased much more rapidly than the general population. New demands for charitable and social purposes are constantly being made upon state funds, but it would seem that any measures for the prevention of insanity which offer hopes of success should receive the substantial financial support of the state.

The attempt has been made to outline some of the preventable causes of mental disease and to indicate, very broadly, possible preventive measures. It seems essential that, notwithstanding the complexity of some of the questions involved, the prevention of mental diseases should be considered in the general advance which is being made against microbic diseases, for it is very closely related to all the other fields of preventive medicine. Recent advances in the field of psychiatry have, upon the whole, given grounds for encouragement, for if the outlook in some directions is not bright the accuracy with which the part played by certain causes has been defined promises much. The fact that it has been definitely determined that there are certain *essential* causes of mental disease, and that some of these essential causes are entirely controllable, makes it imperative that preventive measures be undertaken.

CHAPTER VI

SOME GENERAL CONSIDERATIONS

Sources of Infection.—There are two great sources of the communicable diseases of man, viz.: (1) man himself, and (2) the lower animals. Most of the communicable diseases of man, especially those which occur in epidemic form, are peculiar to man. This is the case with typhoid fever, cholera, leprosy, malaria, yellow fever, syphilis, mumps, measles, scarlet fever, typhus fever, infantile paralysis, smallpox, chickenpox, relapsing fever, dengue, and even tuberculosis in large part. It is quite true that some of these infections may be communicated to the lower animals under experimental conditions, but they do not, as a rule, occur in them under natural conditions. In other words, most of the communicable diseases from which man suffers are specific; the degree of specificity varying slightly with the different infections.

It is, therefore, plain that man is the great source and reservoir of human infections. Man is man's greatest foe in this regard. The fact that most of the communicable diseases must be fought in the light of an infection spread from man to man is one of the most important advances in preventive medicine. This new thought has crystallized out of a mass of work in the sanitary sciences during the past decade, especially from researches upon tuberculosis, typhoid fever, cerebrospinal meningitis, and other communicable diseases. Formerly sanitarians regarded the environment as the main source of infection. We now know that water, soil, air, and food may be the vehicles by which the viruses of the communicable diseases are sometimes transferred—that is, they are media of conveyance rather than sources of infection. Most of the microorganisms causing the communicable diseases of man are frail and soon die in our environment, as in the air, soil, or water. Most of them are obligate pathogens and cannot or do not grow or multiply in our environment.

From the lower animals, particularly the domesticated animals, man contracts a number of infections. Thus we contract rabies from the dog; plague from the rat; glanders from the horse; trichinosis from hogs; anthrax from cattle; malta fever from goats; foot-and-mouth disease from cattle; tuberculosis, in part, from cattle; tapeworms and other animal parasites from the meat of fish, fowl, and mammals. Various skin parasites are also contracted from the lower animals, as ring-

worm from cats, fleas from dogs, etc. The number of these diseases and the extent of their ravages are notably less than those contracted from man himself.

The knowledge that most infections are spread rather directly from man to man brings in all the forces of sociology to that of preventive medicine. The task of preventive medicine is thereby rendered much more difficult from the fact that most infections depend upon the control of man himself. We ruthlessly wage war against insects or against infected food or water. In other words, we can arbitrarily control our environment to a very great extent, but the control of man himself requires the consent of the governed. Thus it is easier to stamp out yellow fever than to control typhoid fever. It is easier to suppress malaria than tuberculosis, rabies than influenza, trichinosis than measles. Cattle appear to be mutely thankful when protected by inoculation against blackleg or anthrax, but man rebels against one of the best of all specifics—vaccination against smallpox. The fact that man is the chief source and reservoir of most of his own infections adds greatly to the scope and difficulties of public health work and often makes the prevention of disease depend upon social changes. In this sense preventive medicine is the true sociology.

Modes of Transference.—The viruses of the communicable diseases may take various routes of transference from man to man or from animal to man. These routes are spoken of as the modes of infection, the modes of transference, or sometimes as the vehicles of infection. Formerly they were spoken of as the "channels of infection," but now we restrict that term to the special channels by which the infection enters the body. Thus the channel of infection in tuberculosis may be the respiratory tract, the digestive system, or the skin; whereas the mode of infection is from tuberculous sputum, either by direct contact or through the air, as in droplet infection, or through milk or some other vehicle.

The modes of transference may be grouped, for convenience, under three general heads: (1) direct, (2) indirect, and (3) through an intermediate host. In the great majority of cases the virus is transferred more or less directly by what is now known as contact infection. In many instances the virus is transferred indirectly through water, food, soil, air, etc. In a large group of diseases the transfer is through an intermediate host which furnishes the growing list of insect-borne diseases.

Contact Infection.—"Contact infection" is a convenient term intended to include a group of circumstances in which infection is spread more or less directly or indirectly from person to person. Contact infection assumes a transfer of quite fresh infective material. Actual contact between the two individuals is not necessary, but the convey-

ance is, nevertheless, pretty close in time and space. Contact infection alone may be responsible for epidemic outbreaks, even in the case of typhoid fever.

The diseases in which contact infection plays a dominant rôle are those in which the virus leaves the body in the discharges from the mouth and nose, as tuberculosis, diphtheria, scarlet fever, measles, influenza, common colds, cerebrospinal meningitis, whooping-cough, mumps, etc. Contact infection also pays a large rôle in diseases in which the virus leaves the body in the fecal and urinary discharges, as in typhoid, cholera, dysentery, and other intestinal infections.

In contact infection the virus may be transferred from man to man directly by actual contact, as in kissing, or more indirectly upon soiled hands, contaminated towels, or infected cups, spoons, toys, remnants of food, and other objects which have recently been mouthed or handled by the infected person. As a matter of fact, the ways by which the infection may be transferred, and still be considered contact infection, are numerous and varied. In every instance, however, the transfer is brought about in pretty close association with the infected person.

Indirect Infection.—A large group of diseases are conveyed indirectly from person to person through the water, food, soil, and occasionally through the air. Diseases may be conveyed great distances by means of food or water; they are never conveyed long distances through the air. In the large majority of the diseases contracted by indirect infection the virus is taken into the system through the mouth and discharged from the body in the feces. The best examples of this class are typhoid fever, cholera, and dysentery. The relation of soil, food, water, air, and our environment is discussed separately.

The insect-borne diseases form a large and important group, which are fully discussed on pages 181 to 274.

Carriers.—By the term "carrier" we understand a person who is harboring a pathogenic microorganism, but who, nevertheless, shows no signs or symptoms of the disease. Thus a person may have diphtheria bacilli in the nose and throat, but, nevertheless, be in good health. The same is true with the pneumococcus, the meningococcus, streptococcus, and many other microorganisms. Persons may have typhoid bacilli, cholera vibrio, or hookworm in their intestinal tract without showing manifestations of these parasites. Furthermore, persons may have plasmodia in their blood or spleen without having clinical malaria, and so on through a long list of infections.

Persons who harbor pathogenic bacteria without showing symptoms are known as "bacillus carriers," those who harbor protozoa are known as "protozoon carriers," etc. Carriers may be acute, chronic, or "temporary"—that is, a person who discharges pathogenic microorganisms a few weeks after convalescence is known as an "acute carrier," one who

continues to harbor the microorganism for months and years is known as a "chronic carrier." A "temporary carrier" is a person in good health who has never had the infection, but who harbors and discharges a pathogenic microorganism for a brief space of time.

The demonstration that many persons are carriers of infection has thrown a new light upon the control of the communicable diseases. With the new facts has come a realization of added difficulties. Carriers can only be detected by painstaking laboratory examinations. When discovered their control is as difficult as it is important. We cannot lightly imprison persons in good health, even though they are a menace to others, especially in the case of bread winners. In some infections there are so many carriers that it would require military rule to carry out such a plan. Fortunately in most cases absolute quarantine is not necessary. Sanitary isolation is sufficient. Thus the danger from a typhoid carrier may be neutralized if the person exercises scrupulous and intelligent cleanliness, and is not allowed to handle food intended for others. Such a person might well be engaged as carpenter, seamstress, or other occupation without endangering his fellowmen.

The fact that carriers exist in a large number of diseases makes their suppression one of great practical difficulty. The cure of carriers is one of the pressing problems in preventive medicine. One hopeful feature of the carrier situation is that their number may be diminished by isolating and diminishing the cases of the corresponding disease. Thus, the number of typhoid carriers falls off sharply as a result of any successful measure directed only against the clinical case. The facts concerning carriers have been discussed separately under each disease in which they occur.

Missed Cases.—By missed cases we understand mild and atypical instances of disease which are not recognized clinically. Almost all diseases vary greatly in severity. Thus we have walking typhoid and ambulant plague. Measles, scarlet fever, yellow fever, influenza, and most other infections may be so mild that they escape notice. Even the patient himself may not know he is sick. These mild cases go to school, ride in street cars, attend theaters, continue at their usual work in crowded factories and other places, handle our food, and thus spread infection. It is now well known that missed cases are a prolific source of spreading the infection of many of the communicable diseases; they form an important factor in preventive medicine.

Channels of Infection.—There are numerous channels by which infection may enter the body. These are usually grouped under three headings: (1) the respiratory tract, (2) the digestive tract, and (3) through the skin. Perhaps 90 per cent. of all infections are taken into the body through the mouth. They reach the mouth in water, food, fingers, dust, and upon the innumerable objects that are sometimes

placed in the mouth. The fact that the great majority of infections are taken by way of the mouth gives scientific direction to personal hygiene. Sanitary habits demand that the hands should be washed before eating, and fingers should be kept away from the mouth and nose, and that no unnecessary objects should be mouthed. All food and drink should be clean or thoroughly cooked. These simple precautions alone would prevent many a case of infection.

"Contagious" and "Infectious."—These are popular terms which lack scientific precision. The words have been used in very diverse senses. A *contagious* disease is one that is readily communicable—in common parlance, "catching." Formerly a contagious disease was considered as one which is caught from another by contact, by the breath, or by effluvia. If contagious diseases are limited to those contracted by direct contact or touch, as the etymology of the word signifies, only syphilis and diseases similarly contracted would be contagious. As a matter of fact, smallpox and measles are types of contagious diseases, as the term is now usually understood.

An *infectious* disease is usually considered as one not conveyed directly and obviously, as in the case of contagion, but indirectly through some hidden influence or medium. In the days when specific febrile diseases were regarded as caused by miasmata and noxious effluvia, the terms "infectious" and "miasmatic" diseases were more or less synonymous. Typhoid fever was often taken as a type of an infectious disease. Malaria was the type of a miasmatic disease.

These distinctions are entirely artificial, and serve no useful purpose. Most of the communicable diseases may be transmitted from the sick to the sound in several ways. Dividing diseases into those which are contagious and those which are infectious entirely leaves out of consideration the important class of insect-borne diseases. The terms contagious and infectious have always lacked scientific precision and have been the source of some confusion. The word "communicable" is a much better term and should be given preference.

A *communicable* disease is one caused by a specific virus transferred from person to person, or from animal to animal, in a great variety of ways. The term "communicable" ignores the mode of transference. There is a great difference in the degree of communicability; some diseases are readily communicable, others transmitted with difficulty. The evidences of communicability are not so obvious in chronic infections, such as tuberculosis, or in diseases with a long period of incubation, such as typhoid fever. The relationship between one case and the next is often far removed in time and space. If tuberculosis were an acute infection like diphtheria it would be regarded popularly as being just as contagious as that disease.

Epidemic, Endemic, Pandemic, and Prosodemic.—A disease is said

to be *epidemic* (epi=in, and demos=people) when it is common to or affecting at the same time a large number of persons in a community. A disease which spreads rapidly and attacks many people at the same time is usually said to be epidemic.

A disease is said to be *endemic* (en=in, demos=people) when it is peculiar to a district or particular locality, or limited to a class of persons. An endemic disease is one which is constantly present to a greater or less degree in any place, as distinguished from an epidemic disease, which prevails widely at some one time or periodically. A *sporadic* (occurring singly) disease is one in which a few scattering cases occur now and then.

Endemic diseases are apt to flare up and become epidemic. Insect-borne diseases are the best examples of endemicity, as their prevalence is strictly limited by the geographic distribution of the intermediate host. Yellow fever has long been endemic in Havana, cholera in India, typhoid fever in Washington, and plague in Tibet.

These terms not only lack precision, but are variously conceived and differently defined. Thus typhoid fever is said to prevail in Boston, but a similar number of cases in Germany would be regarded as an epidemic. For the purposes of maritime quarantine a disease is considered epidemic if there is more than one focus of infection; that is, if several cases occur which have no apparent connection with each other. Strictly, therefore, according to this definition, two cases may constitute an official epidemic and the port would, therefore, be regarded as infected.

It is not feasible to state just how many cases of a disease constitute an epidemic. Ordinarily a few cases of a communicable disease in a village or small town is not regarded as an epidemic; however, five cases of typhoid fever in Podunk (population 1,000) is the equivalent of 5,000 cases in a city of 1,000,000. By the same token, one or two cases in a small village would proportionately constitute an epidemic of unknown magnitude in a metropolis.

"Pandemic" (pan=all, demos=people) is a term used to describe a disease which is more or less epidemic everywhere. Pandemics affect a large number of people in a large number of countries at the same time. Thus there have been four great pandemics of plague, when it spread to the four quarters of the globe. In 1889-90 influenza was pandemic. It is not usual, although quite proper, to regard tuberculosis and typhoid fever as pandemic.

Sedgwick proposes the term *"prosodemic"* (proso=through, demos=people) to take the place of the unsatisfactory word "endemic." Prosodemic suggests the prevalence of a disease which is being communicated from person to person through the community by various means, but especially by contact.

The Management of an Epidemic Campaign.—The first essential for success in the suppression of an epidemic is a knowledge of the epidemiology of the disease. The most important single information from a practical standpoint is a knowledge of the mode of transference of the infection. We do not know the cause of yellow fever; however, yellow fever campaigns have been crowned with success because we know it is transmitted through the bite of a mosquito. We know the cause of cerebrospinal meningitis, but there is still uncertainty concerning its usual mode of transmission, and, therefore, our efforts against this disease have been unavailing. The fact that we know that hookworm disease is transmitted by the larvæ through the skin is of vital importance for the control of this disease. Without this knowledge at least 90 per cent. of our efforts to repress hookworm disease would be wasted. When typhoid fever was regarded as chiefly a water-borne infection only partial success was achieved, because contacts, milk, flies, and other modes of transference of the typhoid bacillus were disregarded.

In case the disease has an intermediate host or the virus is transferred by an insect or other animal, a knowledge of the biology of the animal in question is of prime importance. For example, the habits and habitat of the yellow fever mosquito are quite different from that of the malarial mosquito. A campaign against the rat and flea without an acquaintance with their breeding and feeding places and the best means available to repress or suppress such vermin would be unsuccessful. The same is true in our campaign against tuberculosis with reference to cattle and man; in rabies with reference to dogs and other mammals; in sleeping sickness with reference to the tsetse fly; in Texas fever with reference to the tick; malta fever with reference to the goat; relapsing fever to the bedbug, and typhus fever with reference to the louse.

Authority.—Proper authority is necessary in order to enforce the necessary measures. This authority may come from the municipality, the state, or the federal government. In localized outbreaks, municipal authority is sometimes sufficient. More frequently the wider authority of the state is desirable. In our country it is a recognized principle in law that health laws and regulations belong to the police powers of the individual states. In most instances the general authority of the government must be had, especially as interstate problems are almost always involved in all epidemic outbreaks. The federal authority is limited in health matters by the constitution. It therefore cannot act within a state unless invited to do so by the duly constituted authorities of the state. To send government health officers into a state against the will of the state corresponds to the sending of the regular army into a state to enforce measures against the will of the governor of

that state. Such extreme measures are, therefore, only taken in times of emergency. Occasionally a state, refusing to take necessary action and protect the other states, is quarantined. Thus, when California refused to officially recognize the existence of plague in 1899, the government quarantined the entire state. On account of our dual form of government it is important that the federal government, the state, and the local authorities coöperate in a friendly spirit. Epidemic diseases recognize no geographical boundary, and energetic and coöperative action is usually called for to suppress an outbreak.

It is the common experience of those who have to deal with epidemics that there is usually insufficient authority in law to provide for an emergency. It is, therefore, often necessary to take the bit in the teeth and adopt arbitrary measures which usually have the support of the better element in the community. Advantage may be taken of an epidemic to obtain laws to improve the health organization or the powers of the health officer. In this way an epidemic serves a useful purpose in arousing action.

In the conduct of an epidemic it is very important that all the authority should center in one person. To conduct an epidemic with a board of health or a health committee or a commission of any kind invites failure. It would be just as foolish to have a board of generals to fight a battle. Those who have been through many epidemics realize that it is no figure of speech to compare an epidemic campaign to a battle. It is a fight carried on at high tension, and, although the foe is invisible, it is a battle in every sense of the word.

Ways and Means.—It is impossible to carry on a successful campaign against an epidemic without material resources. An epidemic campaign is expensive and success depends upon generous support. In most of the campaigns against yellow fever, plague, and cholera that have been waged in this country the expense has been borne in part by the government, in part by the municipality or state, and in part by subscriptions from citizens. The government has an epidemic fund appropriated by Congress and which is usually kept at about a million dollars. This fund is available only for plague, yellow fever, and cholera.

Organization.—Headquarters should be organized at a convenient part of the city or the infected area, and headquarters should have all the modern office equipment and transportation facilities necessary for the quick dispatch of business. The city is then divided into sanitary districts. These may correspond to the political wards or the police districts and a subordinate is placed in charge of the work in each district. These districts are known as divisions, and the officer in charge of each division must establish headquarters for the work of that divi-

sion. The actual work is done from division headquarters, under the direction of the chief in charge of the epidemic.

It is also necessary to establish a laboratory in case laboratory diagnosis is necessary for the recognition of cases or carriers, and emergency hospitals and detention barracks must be provided. Few cities have sufficient hospital facilities to meet a sudden emergency. Temporary arrangements must therefore be made. A modern school building makes a very good hospital and may be equipped for the reception of patients at short notice. Various squads must now be organized to carry on the particular work at hand. In the case of yellow fever these will be mosquito brigades; in the case of plague, rat brigades and disinfectors, and in the case of smallpox, vaccinators, etc.

It is frequently desirable, in fact often necessary, to make a house to house inspection throughout the infected district in order to collect certain data, to determine whether cases are being reported or hidden, and to carry out special measures. These house to house canvasses are under the immediate direction of the officer in charge of the sanitary district and should be repeated as often as the occasion may demand.

It is essential that all cases or suspected cases of the disease be promptly reported, for a case of communicable disease known is a case neutralized. It is the missed cases and the hidden cases that are particularly dangerous.

Education.—A campaign of education should be carried on at the same time that the disease is being attacked. The people are keenly alive and hungry for information. Well-worded articles in the newspapers, circulars, pamphlets, lectures, demonstrations, and the other usual methods are available. The education of the community is important in order to obtain coöperation, for it is a handicap to fight an epidemic without the active support of the people. While the first duty of the officer in charge is to allay panic and calm the unreasonable fears of the stricken community, the opposite extreme must be avoided. A healthy fear of the disease is one of the best instruments in the armamentarium of the sanitarian. It is almost hopeless to make progress against disease where the people supinely accept the conditions. Thus, if the people of the United States feared typhoid fever as they do yellow fever, it would soon diminish to the vanishing point.

QUARANTINE

The word "quarantine" is derived from the Italian word "quarante," meaning forty. Its present-day meaning dates from the middle ages when Venice and other Hanseatic cities detained arriving ships with cases of pestilence aboard for a period of forty days. This was the first systematic application of maritime quarantine, although from the earliest times lepers were segregated or quarantined. To-day we have

many kinds of quarantine: maritime quarantine, interstate quarantine, house quarantine, cattle quarantine, yellow fever quarantine, shotgun quarantine, etc.

The dominating principle in modern quarantine is that it must be a sieve or filter and not a dam. All quarantines based upon the principle of the Chinese wall are doomed to fail. The object of quarantine is, then, to destroy, detain, or isolate infection with the least possible hindrance to trade and travel. The art consists in regulating the openings in the quarantine sieve so as to hold back certain infections, but permit all else to pass. Maritime quarantine may be regarded as a coast defense against exotic pestilence, a defense which guards against an invisible foe ofttimes more damaging than hostile armies and navies. The cure for quarantine is sanitation.

If all communities, especially seaports, were to place their cities in the best sanitary condition in accordance with the teachings of modern science, there would be little danger of disease spreading to epidemic proportions and very little need of maritime quarantine. If the ports in our southern littoral would free themselves of the Stegomyia mosquito they could laugh at yellow fever. A city containing few rats could not have an epidemic of plague. A port supplied with a pure, well-protected water supply need not fear a water-borne epidemic of cholera. A thoroughly vaccinated community runs no hazard from smallpox. Typhus fever could not spread in a community with cleanly personal habits, that is, one free from lice and other vermin.

Maritime Quarantine.—Maritime quarantine in this country is enforced only against six diseases, viz., cholera, yellow fever, plague, typhus fever, smallpox, and leprosy. We do not quarantine against typhoid fever, tuberculosis, measles, and other infections which are not greatly feared and which are constantly with us. Infections of a non-quarantinable nature, such as scarlet fever, measles, etc., arriving at a port are permitted to enter, but must then comply with the local laws and regulations.

The period of detention is based upon the usual period of incubation for each disease and is as follows:

Cholera	5 days.
Yellow fever	5, sometimes 6 days.
Plague	7 days.
Typhus fever	12 days.
Smallpox	14 days.
Leprosy	not admitted.

The time of detention is usually counted from the completion of disinfection or at least from the last possible exposure to the infection. This is usually not a very difficult matter for the quarantine officer to decide, but in case of doubt the public is given the benefit.

No communication is permitted with a vessel in quarantine excepting under supervision of the quarantine officer; that is, no one is allowed to board the vessel or leave it, and nothing is allowed to be thrown overboard, taken ashore, or brought on board without the express permission of the quarantine officer. These restrictions apply alike to foods and to merchandise of all kinds.

The vessel itself may be disinfected and furnished with a fresh crew and released from quarantine while the passengers and crew are detained in suitable barracks. Vessels trading with infected ports should carry immune crews; that is, persons who have either had the disease or have been rendered actively immune through one of the vaccines or viruses.

When a quarantinable disease breaks out on board a vessel it is of practical importance for the quarantine officer to determine whether the infection was contracted on board the vessel or on land. In the first case the vessel must be regarded as infected and the measures used for its purification are much more exacting than in the second case. Thus, if plague breaks out within five days from the time a vessel leaves an infected port, and no other case occurs, it is exceedingly probable that the patient contracted his disease ashore and was in the period of incubation when he came on board. If, however, plague breaks out after five days, and especially if secondary cases occur, it is evident that the ship itself is infected. The same reasoning applies to yellow fever and the other communicable diseases.

The measures taken at quarantine to keep out these diseases depend upon an accurate knowledge of their cause and mode of transmission. Briefly summarized, the measures applicable in each case are as follows:

Cholera.—Cases are removed from the vessel and isolated and that part of the vessel and the objects exposed are disinfected—formaldehyde for cabins, sulphur dioxid for the hold, bichlorid solution for surfaces, steam for fabrics and clothing. A search is made for bacillus carriers and a bacteriological examination is made of all cases of diarrhea. Special attention is given to the water supply, food, and flies. After the sick are isolated the remainder are segregated in small groups. Those especially exposed are first bathed and their body clothing disinfected before they are sent to the detention barracks. In case of cholera arrangements should be perfected for the disinfection of the dejecta. Baggage which has been exposed is disinfected by an appropriate method, but as there is little danger in the cargo, especially if it consist of new manufactured merchandise, this may be passed without special treatment.

If a vessel has taken water ballast at an infected port it is required to empty the same at sea and replace the presumably infected water with sea water. If this has been neglected the vessel must return to

sea past the three-mile limit for this purpose. The water and the water tanks may be rendered safe by the use of chlorinated lime.

The period of detention in the case of cholera is five days.

Smallpox.—Ordinarily those who have had smallpox or who have had a recent successful vaccination are not detained. All others must submit to vaccination. Persons declining vaccination are detained for the full period of 14 days before they are released. As a rule, it is not necessary to detain cabin passengers because there is smallpox in the steerage, or to detain the firemen because there is smallpox among the stewards. Vessels arriving with smallpox on board on which the cases have been properly isolated, personnel vaccinated, and other sufficient precautions taken to prevent the spread of the disease, need not be quarantined further than the removal of the sick, the disinfection of compartments, baggage, and objects that have been exposed to the liability of infection.

Plague.—Passengers and crew from plague-infected ports are carefully inspected at quarantine. The temperature of each person should be taken and it is desirable to make special examinations for bubos. A careful search is made for cases of *Pestis minor,* and the pneumonic form of the disease must also be kept in mind. The period of detention in the case of plague is 7 days. The sick are isolated in the hospital and the remainder segregated in small groups. All persons exposed to the infection are bathed and their body clothing disinfected.

Rats and fleas on the vessel must be killed and burned. Usually sulphur dioxid is used; sometimes hydrocyanic acid gas or carbon monoxid.

Special precautions must be taken to prevent the escape of rats. Vessels quarantined on account of plague should be anchored at sufficient distances from shore to discourage rats swimming to the land. If the vessel ties up to the dock, the hawsers must be guarded with inverted cones or balls of tar in order to stop rats reaching the shore along these lines. Gangplanks must be taken in before dark, and, as rats are nocturnal in their habits, a searchlight will help to deter them from leaving the ship. Nothing should be thrown overboard, not even deck sweepings; these should be burned, but not in the galley.

A plague-infected ship is given a simultaneous disinfection with sulphur and the cargo is removed by a special procedure. After sulphuring, the cargo is removed piece by piece to lighters, each article being examined as it swings overboard for rat nests. This work goes on during the day, while the empty cargo spaces are fumigated with sulphur during the night in preparation for the next day's unloading.

Special precautions must also be taken at foreign plague ports to prevent the ingress of rats and also to prevent unnecessary human communication with infected areas. All vessels trading regularly with

plague ports should carry an approved type of sulphur furnace, such as the Clayton apparatus, to use during the voyage, in order to kill rats that may be on board. Such vessels should have an immune crew; that is, persons who have either had the disease or have been protected with Haffkine's prophylactic.

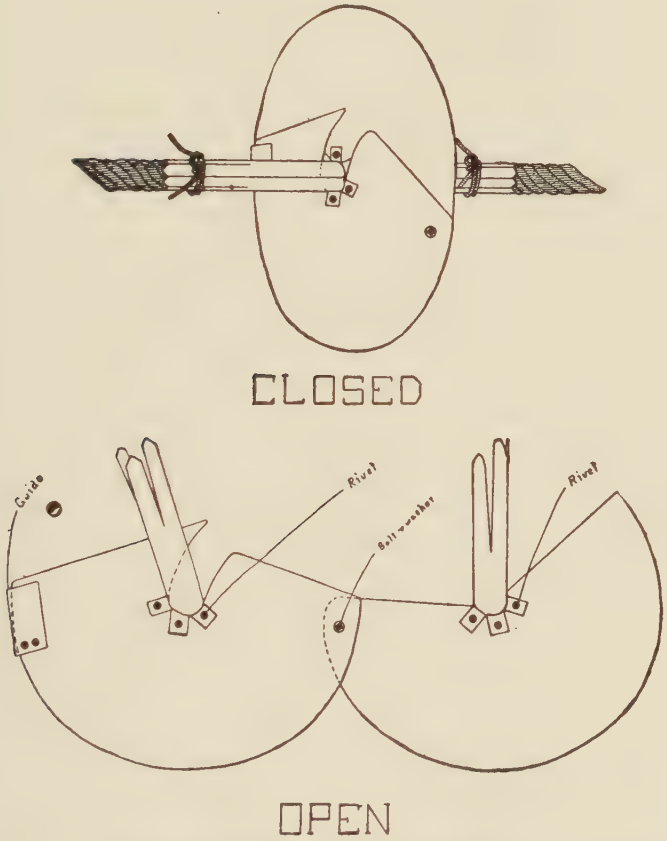


FIG. 47.—A DEVICE FOR PREVENTING RATS TRAVELING ALONG HAWSERS.

Yellow Fever.—Vessels arriving at an infectible port from an infected port are fumigated and detained five days as a precautionary measure during the yellow fever season, even though there is no evidence of sickness on board. The yellow fever season usually extends from May 1 until October 1. The infectible ports are those situated upon the Atlantic seacoast south of the Chesapeake and those on the Gulf of Mexico.

Five days covers the period of incubation of most cases of yellow

fever and is sufficient as a precautionary measure, but in special instances, as, for example, if a case of yellow fever has occurred on board the vessel, then the detention is six days following fumigation. The sick are isolated by the use of mosquito screens. Patients with yellow fever should not be moved if this involves exertion or excitement, which may aggravate the disease.

The vessel is fumigated with an insecticidal substance, preferably SO_2, in order to kill the *Stegomyia calopus.* A search is made for breeding places, such as water casks, fire buckets, and other collections of fresh water where the Stegomyia larvæ and pupæ may develop. The disinfection of baggage and fomites is no longer practiced in the case of yellow fever. Experience has shown that wooden vessels are more apt to convey yellow fever than iron vessels. This is because wooden vessels carry water casks, which are the favorite breeding places for the mosquito, while iron vessels store their drinking water in tight compartments deep in the hold, inaccessible to mosquitoes. Vessels plying between infected and infectible ports should carry immune crews.

Typhus Fever.—The period of detention for typhus fever is 12 days. If a case of typhus fever occurs upon a vessel and has been properly isolated, and the vessel is in good sanitary condition, there is practically no danger of its spread, the case may be removed, disinfection practiced (insecticides), and the vessel, passengers, and crew permitted to proceed. But, if the case has not been isolated, or if the disease has spread from one person to another upon the vessel, or if the ship is infested with vermin and is otherwise in an unsanitary condition, those exposed are detained in quarantine until the period of incubation has elapsed. Quarantine procedures in the case of typhus fever are now focused entirely upon the louse, which is the carrier of the infection.

Leprosy.—An alien leper is not allowed to land. The law requires the vessel on which he arrives to take him back again. It is unconstitutional to forbid the landing of an American leper, but as soon as he lands he comes under the laws of the city or state in which he finds himself. Alien lepers are detained at the quarantine station and placed aboard again when the vessel is outward bound.

Quarantine Procedures.—All vessels arriving at any port in the United States from a foreign port are considered to be in quarantine until they are given free practique. The practique is a certificate signed by the quarantine officer to the effect that the vessel and all on board are free from quarantinable disease, or the danger of conveying the same. In other words, free practique is a permit issued by the quarantine officer which the master of the vessel must present to the collector of the port in order that his vessel may be admitted to entry.

Vessels in quarantine are required to fly a yellow flag (letter "Q" of the International Code) from the foremast. The quarantine officer

boards the vessel usually upon the starboard side and examines the bill of health, the ship itself, the passengers, the crew, as well as the manifests of cargo, and sometimes the food and water supplies, etc. Vessels arriving after sundown must wait until sunrise for this inspection; the time and details, however, vary greatly and depend upon circumstances. Thus, at the port of Boston, there is no more need to examine vessels bringing residents of London or Paris than there would be to examine a trainload of passengers from New York.

The detection of infection on board a vessel requires knowledge, tact, and sometimes a detective instinct on the part of the quarantine officer. Where one of the communicable diseases is suspected the temperature of every person on board should be taken. Sometimes special examinations, as for bubos in the case of plague, are necessary. As a rule, all hands are mustered at a designated place on board the ship and then passed in review, one by one, before the examining physician; the number of persons are counted and compared with the ship's papers; each person is critically scrutinized for evidence of disease, and suspects are placed aside for more careful examination later. The clinical records of the ship's surgeon are inspected with special reference to the diagnosis of those who have received medical care during the voyage. The manifest of cargo is examined for second-hand goods, upholstered furniture, bedding, hides, hair, or other objects that may require disinfection. Finally, the ship itself is inspected, attention being given especially to the forecastle, steerage quarters, the galley, etc.

The Bill of Health.—The United States Bill of Health is a document issued by our consul at the port of departure to the master of the vessel. The Bill of Health contains a complete description of the vessel, the number of officers, crew, and passengers (cabin and steerage), its sanitary history, and the sources and wholesomeness of water, food supply, etc. Finally, it contains a statement giving the number of cases and deaths from yellow fever, cholera, smallpox, typhus fever, plague, and leprosy at the port of departure during the two weeks preceding the departure of the vessel. The American Bill of Health, which is a formidable document, must be obtained by the master of the vessel in duplicate; one copy is destined for the collector of customs at the point of entry and the other for the quarantine officer.

The Bill of Health is a consular document (State Department) at the port of departure, but becomes a customs paper (Treasury Department) at the port of entry. Vessels arriving at any port in the United States or its dependencies from a foreign port without this official Bill of Health in duplicate are subject to a fine of $5,000. Before the days of telegraphy the Bill of Health was an important document and often gave the quarantine officer the first information of pestilential disease abroad. The quarantine officer must now keep himself informed not

only of the health conditions of the port of departure, but of the places from which the passengers and crew are recruited.

There are many kinds of bills of health; each country has a form of its own. Formerly a bill of health was simply a statement that the port of departure was or was not free of pestilential disease; that is, the bill of health was either "clean" or "foul." The American Bill of Health gives much more valuable information in detail. The only bill of health that is of service to the vessel upon arrival is the American Bill of Health, although several bills of health may be issued to the vessel at the port of departure. Thus, a British vessel leaving the port of Rio de Janeiro takes three bills of health, one from the British consul, required by the British admiralty laws, another from the Brazilian authorities, which is a clearance paper, and the third from the American consul, which is the only one of service upon reaching a port in the United States.

The Equipment of a Quarantine Station.—The equipment of a quarantine station consists of boarding vessels, such as tugs, launches, and rowboats; of an inspection place where passengers, crew, and suspects may be examined (the facilities on board the ship are usually inadequate for this purpose); of disinfecting apparatus for the use of steam, sulphur dioxid, formaldehyde, and insecticides; shower baths; detention barracks for steerage, intermediate, and cabin passengers, as well as the crew of the vessel; isolation wards in which cases of the quarantinable diseases may be cared for, and special wards where suspects or non-contagious cases may receive treatment. A well-equipped quarantine station further needs dining-rooms and kitchens for the various groups detained; quarters for the quarantine officers and help; a wharf and boat house, and some provisions for recreation of those in quarantine to dispel the *ennui* of the isolation. Finally, a crematory, a steam laundry, and special arrangements for the disposal of sewage and garbage are essential.

A laboratory is an essential feature of a modern quarantine station. It is necessary in order to make diagnoses and to recognize bacillus carriers, etc. In other words, a quarantine station, on account of its importance and isolation, must be a well-equipped and self-supporting community.

Qualifications of the Quarantine Officer.—The quarantine officer must be a good diagnostician. He should have a special acquaintance with the diseases against which he stands monitor. Further, he must be familiar with the modes of spread of the quarantinable diseases and must know the value and limitations of the germicidal agents and insecticides he uses. Finally, he must be familiar with matters nautical, and have an extensive knowledge of geography. It is the duty of the quarantine officer to keep posted as to the sanitary conditions

of all countries, especially the towns and places having commerce with his port.

Disinfection of Ships.—The disinfection of a vessel does not differ materially from the disinfection of houses and rooms. It should not, however, be attempted by one not familiar with the intricacies of marine architecture and matters nautical, for many special conditions are met with on board ship that are very different from those found on shore. While the principles of disinfecting as applied to a vessel present nothing unusual, the application of these principles calls for much ingenuity and the keenest vigilance on the part of the disinfector.

It is important to enlist the sympathies of those on board with the necessity of disinfection, for the successful accomplishment of the purification of the vessel may be materially helped by the cheerful coöperation of the passengers and crew; otherwise the difficulties of the problem are greatly magnified.

Formerly a distinction was made between the methods of disinfecting a wooden and an iron vessel. This arose from the fact that almost all wooden vessels have some rotten and spongy wood, especially about the forefoot and bilge. There are also many more cracks and open joints about a wooden ship than a metal one which afford lodgment for organic matter. In addition to this, a wooden hull is always damper than an iron hull, for almost all wooden vessels leak more or less. It was formerly believed that the microorganisms of disease were apt to become deeply lodged in the moist dirt and organic matter of the many crevices, but we now know that this is largely theoretical.

A vessel is rarely so badly infected that it needs a disinfection throughout. Just what portion of the vessel and its contents requires treatment is often a very difficult problem to solve. There is no more reason to fumigate the hold of a vessel because smallpox appeared in the cabin or steerage than there would be to disinfect the basement and subbasement of a tenement house because a case appeared in one of the upper stories of the building. When a communicable disease occurs on board a vessel the infection may be confined to one or two compartments or to a limited area quite as successfully as this may be done in buildings on shore. "In case of doubt, disinfect," is not a bad rule for the quarantine officer to follow in his practical dealings with ships. For, after all, the measures which must be taken are greatly in excess of the absolute requirements.

Much may be learned by a thorough inspection of the vessel. To be sure, we cannot see the germs with our unaided vision, but we can see the dirt and moisture and other conditions which favor their life and virulence and can discover the feeding and breeding places for vermin.

It is, therefore, the duty of the quarantine officer to require a very

thorough mechanical cleansing of all parts of the ship which, in his judgment, require it. This matter is dwelt upon because filth and vermin are conditions too frequently met with on the sea and one of great importance to communities and nations.

While the general methods of treating vessels are the same for most of the bacterial infections, special methods are called for with each disease. For example, in cholera particular attention must be paid to the water and food supply; for plague the destruction of rats and fleas is of prime importance; for yellow fever attention must be directed against the mosquito; for smallpox vaccination and the usual disinfection of the living apartments, clothing, bedding, and the like are required, while for typhus fever the warfare must be waged against lice.

The disinfection of a large vessel cannot effectively be done without all the modern contrivances of a well-equipped quarantine station. A rowboat and launch or a small sailing craft may be disinfected with a tub of bichlorid solution, but good work cannot be accomplished on a large vessel by the use of makeshifts.

Before the disinfection of a vessel is commenced it should be brought alongside the pier or barge containing the necessary apparatus. All the passengers are then to be taken off and all the crew, only excepting the few who are necessary for the safety of the vessel and those who are to help in the disinfection. The quartermaster, the boatswain, and the carpenter are very useful hands to aid in the process on account of their practical knowledge of the individual peculiarities of the construction of the vessel and their intelligence in carrying out directions with faithfulness.

When the personnel have left the vessel all their effects are removed and disinfected, if necessary, in accordance with the methods outlined for objects of that class. Baggage, bedding, and other objects, no matter what their character, after disinfection should not be returned on board until the treatment of the vessel itself is finished. This injunction applies, of course, equally well to persons. In fact, no one should be allowed on the vessel except those actually engaged in the work, who, as far as practicable, should be immune and should wear suitable garments. All the bedding, bed clothing, hangings, floor runners, and other fabrics that have been exposed to infection must now be removed to the steam chamber. Especial care must be taken to obtain all the used and soiled linen, which is usually kept in special compartments called the "dirty linen lockers," which are usually under the care of one of the stewards. For some reason there is a dislike to disclose the presence of this soiled wash to the quarantine officer.

After all the objects needing disinfection by special process have been removed, attention is then directed to the vessel itself. The various compartments of the vessel may be disinfected by any one of the

methods described under Room Disinfection, formaldehyde being the choice of the gases and bichlorid of mercury (1-1,000) being the most suitable solution for the treatment of walls, floors, etc.

The bichlorid solution, which is sometimes used for flushing the forecastle, the steerage compartments, and quarters for petty officers, etc., may be applied with a force-pump or by means of mops and buckets. In applying the disinfection solution with a hose begin at one end of the deck ceiling and systematically flood every inch of surface, coming down the walls, and finally the floor.

In disinfecting large vessels it is well to start forward with the forecastle and work aft systematically, first on the starboard, then on the port side, taking care to require every door to be unlocked and trusting only to a personal inspection concerning its contents and uses. There are certain places, such as the lamp-room, the paint locker, the sail locker, the chain locker, the carpenter shop, and chart room, the pilot house, the engine and boiler rooms, and the machinery, that are rarely infected, and, as a rule, need no treatment. Special care, however, must be given to the sick bay and any apartment in which a patient was cared for, and all living apartments, including the steerage.

The water closets on board ship should be thoroughly cleansed and flushed with water and may be disinfected with chlorinated lime or carbolic acid. They may also be hosed with the bichlorid solution while that is being applied. In sailing vessels of the older type the forepeak needs similar treatment.

The hold rarely needs treatment on account of bacterial infection. About the best way to disinfect the holds of vessels is by sulphur fumigation or by a solution of corrosive sublimate applied with a hose. The bilge may be flushed with carbolic solution or chlorinated lime and then pumped out. When the hold is fumigated with sulphur, this may be burned in iron pots set in pans of water. The pot should be placed in an elevated position either on piles of ballast or on the 'tween decks. In leading sulphur fumes into the holds from a sulphur furnace it is considered best to lead the pipes down the hatch-well toward the bottom of the hold, so that the apartment may fill up with the fumes from the bottom, displacing the air above. For this reason openings above for the escape of the air must be provided. This is best managed by leaving one or two of the ventilators open, or part of the hatch, and after the gas has begun to escape in some quantity to close up tight.

The amount of sulphur to be burned may readily be computed from the tonnage of the vessel. A registered ton is 100 cubic feet. Count half a pound for each ton, which will make the necessary five pounds per 1,000 cubic feet. The gross tonnage of a vessel indicates her actual cubic capacity. The net tonnage gives the capacity of her cargo-carrying space. The difference between the two will give the capacity

of the spaces devoted to the engines, machinery, living apartments, storerooms, etc. In sailing vessels and in freighters the net tonnage may be taken as the cubic capacity of the hold. In estimating freight 40 cubic feet of merchandise is considered a ton, provided the bulk does not weigh more than 2,000 pounds. This ton, used as a commercial unit for freight charges, must not be confused with the registered tonnage based upon the measurement of the vessel.

In fumigating vessels for yellow fever, plague, and other insect- or animal-borne diseases, the fumigation should be simultaneous in all parts of the vessel. Following this, special rooms and apartments may be given individual treatment, depending upon circumstances.

The empty compartments of an iron steamer may be disinfected by steam, provided it is above the water line. The compartments of such vessels usually have steam pipes for use in case of fire. Clothing and other fabrics may also be disinfected by steam, by exposing them in the compartment.

The water tanks and casks of vessels sometimes need special treatment. The water may be infected with cholera, typhoid, dysentery, or other water-borne infection. The water may be disinfected *in situ* by the addition of chlorinated lime, using an amount sufficient to make a one per cent. solution. This should stand at least 24 hours before it is pumped out.

Water casks on sailing vessels are very apt to be breeding places for mosquitoes. These should be emptied and cleansed. The water containing the larvæ may be spilled overboard, as neither the anopheles nor the stegomyia may develop in salt water, otherwise the larvæ should first be destroyed.

For the destruction and treatment of rats, etc., on vessels see pages 245 and 252.

Cargo.—As a rule, the cargo of a vessel infected with pestilential disease needs no disinfection. Individual articles of the cargo, such as rags, household goods, second-hand articles, or food products, from infected localities may need treatment. New articles of merchandise or new manufactured goods seldom carry infection.

In the case of plague the cargo may need special treatment on account of rats (see page 324).

Ballast.—Vessels bring two kinds of ballast: (1) water, (2) solid. Solid ballast consists of the greatest variety of substances. The kind which is most objectionable from the standpoint of the health officer is called "sand" by the captain, but an inspection of this sand will discover the fact that it often consists largely of street sweepings and rubbish from the port from which the vessel hails. Such ballast should not be unloaded on the city front, especially if it comes from an infected district. Ballast consisting of clean, hard rock or sand from the beach

is not apt to carry infection of any kind, and usually needs no attention from the quarantine officer.

Modern vessels all use water ballast. The tanks may be filled from a river, fresh water lake, or other body where cholera, typhoid, or dysentery prevails. It is a rule in quarantine practice to require vessels in fresh water ballast from cholera-infected districts to return to the open sea, where the ballast tanks are pumped out and refilled with salt water, provided this has not been done on the high seas. Before the water is pumped out it should be treated with chlorinated lime.

Foreign Inspection Service.—To aid the quarantine officer every American consul is required to report regularly certain facts concerning the presence and progress of epidemic diseases. Medical officers of the government are also stationed at various countries in order to supervise the sanitary condition of vessels, their cargo, and passengers leaving for the United States. This may be called preventive quarantine, for it is a distinct help in keeping out infection and facilitates trade and travel. Thus, in Italy, during the cholera times, an officer of the Public Health and Marine Hospital Service stationed at Naples successfully kept that disease off vessels sailing from Naples to the United States, whereas vessels sailing from Naples to other ports and without sanitary supervision carried cholera in several instances.

National versus State Quarantine.—All the maritime quarantines in this country are now controlled by the national government, excepting the ports of Boston and New York. At Boston the maritime quarantine is in charge of the city health authorities, and at New York it is a state institution. At a few other ports a local quarantine is maintained in addition to the national service. The federal quarantine service is administered by the Public Health Service, a bureau in the Treasury Department.

It is evident that maritime quarantine should be administered uniformly so as not to prejudice or favor the commerce of a port. Not only is uniformity insured by a central service, but there is a decided gain in efficiency for obvious reasons. Maritime quarantine deals mainly with foreign shipping. The Constitution reserves for the federal government the right of treating with foreign powers; from this point, therefore, maritime quarantine is mainly a function of the federal government.

Interstate Quarantine.—In accordance with our Constitution the federal government has limited power within the state, but has practically unlimited authority to prevent the spread of infection from one state or territory, or the District of Columbia, to another state or territory, or the District of Columbia. Interstate quarantine involves interstate travel and commerce; the pollution of streams flowing through more than one state; railroad and steamboat sanitation, and all similar

questions. Congress has passed a comprehensive act, Section III of the Act of February 15, 1893, authorizing the Public Health and Marine Hospital Service to enforce interstate quarantines in the case of contagious and infectious diseases. The regulations, however, prepared under this act comprehend only the six quarantinable diseases, and have only occasionally been enforced in the case of yellow fever, cholera, or plague. There are no interstate regulations concerning typhoid fever, tuberculosis, measles, and other non-quarantinable diseases. It is evident that this is one of the important phases in which government activity can accomplish especial good; for, while the government has limited power within the state, it has practically unlimited authority so far as interstate relations are concerned. Widespread diseases will never be adequately controlled by the local authorities without the cooperation of the government. It is evident that, if one state should rid itself of typhoid fever, measles, or tuberculosis, it would soon become reinfected from the neighboring states. Interstate sanitation is one of the burning questions needing vigorous action and cannot be adequately enforced without extending the scope and powers of the present federal health authorities.

ISOLATION

In theory isolation is the most perfect single method to check the spread of a communicable disease. The results in practice, however, have been somewhat disappointing on account of unusual difficulties. The statement has frequently been made, especially with reference to typhoid fever, that if all the cases could be isolated (which includes the disinfection of the discharges) we would soon see an end of the infection. We now know that this statement is not true, on account of the bacillus carriers and the mild and unrecognized or "missed" cases. Because the isolation of the reported cases represents only a portion of all the foci of infection and, therefore, at best could not in itself control an epidemic disease, discredit has been thrown upon this procedure, which is one of the essential features of all systems of quarantine. As a matter of fact, it has been shown that in certain diseases, like measles, which is communicable for three days or more before the nature of the disease is recognized, isolation has practically no influence in diminishing the prevalence of this widespread infection. It is true ordinarily that a case of measles does most harm before it is isolated; nevertheless, this is no reason why it should be permitted to further endanger the community. The value of isolation is also diminished by the prevalence of carriers. In fact, its practical usefulness in a given infection is inversely proportional to the number of carriers.

If each case isolated prevents on the average only one other fresh infection, there would still be justification sufficient to continue the

practice. As a matter of fact, the practical value of isolation varies with each disease, depending upon the degree of its communicability, the time when it is communicable, the promptness by which it may be recognized, the modes by which it is transferred, the existence of latent infections, missed cases, carriers, and other factors which influence the spread of the infection.

The degree of isolation varies markedly with the different infections. A case of yellow fever may be isolated under a mosquito screen, and a case of diphtheria or scarlet fever may be effectively isolated in a bed in a general ward, provided intelligent and painstaking care is exercised to destroy the infection as it leaves the body. Isolation of the more readily communicable diseases, as smallpox and measles, should be much more absolute. Typhoid bacillus carriers need not be imprisoned. It is sufficient to limit their activities, especially to prevent their occupation in kitchens, dairies, or about foodstuffs. There is no good reason to isolate a consumptive or leper without open lesions—that is, cases in which the bacilli are imprisoned in the tissues and not discharged into the environment. A careful consumptive or leper may be allowed a wide latitude. On the other hand, isolation in chronic infections, such as tuberculosis and leprosy, with open lesions is the most helpful and at the same time the most difficult single procedure we have to control their spread. The careless, indigent, ignorant, or helpless consumptive is a public menace that needs energetic and sometimes arbitrary isolation.

Isolation may most readily and effectively be carried out in hospitals or sanatoria. Proper isolation in the home requires a special room or rooms, intelligent nursing, appliances for disinfection, etc., a combination often difficult to arrange. House quarantine varies with the different diseases. To carry it out rigorously in all cases and under all conditions is folly. Different diseases need different procedures. Sometimes it is sufficient simply to placard the house as a warning. At other times it may be necessary to station sanitary guards about the premises to enforce the quarantine. The imperfections of strict isolation by the "shutting in of houses" are graphically described in Defoe's "Journal of the Plague Year."

Isolation camps or temporary barracks in times of epidemics are effective measures in checking the spread of some infections. This method has proved effective in actual practice in the case of smallpox, yellow fever, plague, cholera, and other diseases.

It often becomes a difficult question to determine whether the well members of a household should also be quarantined—especially whether the well children should be permitted to attend school. This perplexing question must be decided for each disease separately, and the decision in each disease is sometimes modified by attending factors. Usually

the other children in the family in the case of scarlet fever are excluded from school for four weeks from the beginning of the last case. In most cities the same rule holds for diphtheria, although here we are able to determine whether the children are bacillus carriers or not. At least two negative cultures from the nose and throat should be required before such children are allowed freely to mingle with other children. The principal factors which determine whether the well children in a family shall be permitted to attend school or not in any particular infection rest upon our knowledge as to whether the disease is conveyed by a third person and the frequency of bacillus carrying and missed cases.

Isolation becomes one of our most valuable public health measures when communicable diseases affect persons working about milk, meat, and other foods capable of conveying infection.

One of the practical objections to isolation and one reason that it meets with so much opposition from the public is that the compensation of the wage earner ceases through no fault of his own. It is evidently unjust to practically imprison and thus seriously punish a member of the community, not for his own good but for the good of the community, because he or some member of his family has contracted an infection, perhaps through some fault of the community itself. It is, therefore, reasonable and just that wage earners and others should be compensated and their personal interests safeguarded during enforced isolation.

Isolation only reduces to a moderate degree the prevalence of disease. The limitations of this valuable procedure are now well understood. With improved methods of diagnosis and increased knowledge of the methods of spread of disease, isolation will be made increasingly effective. Every case isolated is a focus of infection neutralized. Although not as satisfactory in practice as it is in theory, isolation will ever remain one of the chief administrative procedures for the control of the communicable diseases.

SECTION II

IMMUNITY, HEREDITY, AND EUGENICS

CHAPTER I

IMMUNITY

Immunity or resistance to disease is the very foundation of preventive medicine. It is the overshadowing factor in hygiene. In this sense we use the term "hygiene" to include the care of the person, in contradistinction to "sanitation," which deals with the environment. There is no sharp line of demarcation—we speak of hygiene of the teeth, of sleep, of bathing, of exercise, or food and drink, and of those conditions which are more or less intimately associated with the body; we speak of the sanitation of the home, of schools, of cities, of farms; sanitary science considers the air, soil, climate, and our surroundings as they affect health. Sanitation, then, is largely impersonal; hygiene is personal, and, as far as the prevention of disease is concerned, one of the most important factors in hygiene is immunity.

The word "immunity" is a very old term—we still speak of immunity to crime,[1] but it is only of late years that we are beginning to understand the mechanism by which the body protects itself against infection. The advances have been so rapid that these studies may now be grouped into a separate science known as Immunology.

Immunity is a function of all living beings (animals and plants), and in its widest form is one of the fundamental properties of life. Thus, as long as we are alive the colon bacillus in our intestinal tract and the spores of the hay bacillus on our skins do us no harm, but the moment we die, and ofttimes shortly before death,[2] these and other bacteria invade our tissues and disintegrate them.

Immunity may be defined as the power which certain living organisms possess of resisting infections. Immunity is the contrary condition to susceptibility. Hypersusceptibility is a special state of an exag-

[1] We may speak of immunity "from" a disease, "to" a disease, and "against" a disease.

[2] Terminal infections.

gerated power of reaction and will be discussed separately under anaphylaxis or allergie. The word resistance has practically the same signification as immunity. The term "tolerance" is commonly used to describe a limited form of immunity usually acquired by the repeated use of alkaloids, alcohol, and other poisons of comparatively simple chemical structure. While a high degree of tolerance may be acquired to such substances, a true immunity in the sense in which the term is now used is never produced. In the case of tolerance, antibodies are not found in the blood. For the most part true immunity is produced against colloidal substances, while tolerance is largely limited to the crystalloids; this distinction, however, is not absolute.

There are all gradations and various kinds of immunity. It varies in degree from the weakest appreciable amount to an almost absolute protection. It also varies greatly in duration—from the briefest period to a life span. Immunity, therefore, is a relative term. It may be natural or acquired, active or passive, local or general, pure or mixed, specific or general, family or racial, brief or lasting, strong or weak, etc.

Immunity is a function which is not limited to man and other members of the animal kingdom. It is common throughout the vegetable kingdom. We are indebted to Welch for the thought that the bacteria themselves also have this fundamental property of life. Thus, man is susceptible to the tubercle bacillus because the tubercle bacillus is immune to man; on the other hand, man is immune to the hay bacillus because the hay bacillus is susceptible to man. In this sense a microorganism is called pathogenic or non-pathogenic, depending upon whether it harms or is favored by its host. This is the relation between seed and soil. A fertile soil is susceptible; a barren soil is immune. The seed in the first instance may be pathogenic; in the second non-pathogenic. The host is able to resist the intrusion and growth of the non-pathogenic microorganisms and protect itself against harm through its mechanism of immunity. If the protecting devices are insufficient to guard against attack, the germs multiply, produce poisonous substances, or harm the host in other ways. The reason that the same microorganism may be pathogenic for one host and harmless for another depends upon the presence or lack of immunity. The virulence of a microorganism is an expression of the intensity of the relation between the seed and the soil. Virulence may be strengthened or attenuated either by increasing or decreasing the resistance of the host or by increasing or decreasing the resistance of the microbe.

Mechanism of Immunity—Theories of Immunity.—It is now quite evident that the mechanism of immunity varies in different infections and, to a certain extent, even in the same infection under different conditions. It must further be admitted that we are still in ignorance of

the mechanism by which the body protects itself against many diseased states.

Historically considered, immunology as a science dates back scarcely 30 years. Many primitive people attempted to immunize themselves in a crude sort of way, but with methods now recognized as essentially sound. Thus, South African tribes tried to protect themselves against snake bites by using a mixture of snake venom and gum; the Moors immunized cattle to pleural pneumonia by placing some of the virus under the skin of the animal. The inoculation against smallpox used from time immemorial, and vaccination with cowpox introduced by Jenner in 1798, are examples of the first practical use of specific methods in the history of immunity.

Pasteur was greatly influenced by Jenner's demonstration that a mild form of a disease protects against the severe form. Pasteur expanded the fact taught by Jenner into a general principle. Practically all of Pasteur's work in immunity that bore practical fruit, such as vaccinations against chicken cholera, anthrax, and rabies, is based upon this guiding principle.

Pasteur in 1888 expounded his "exhaustion" theory, which was the first attempt at a scientific explanation of immunity. Pasteur was a chemist and his theory was a simple chemical conception, largely based upon his work upon the fermentation of sugar with yeasts. He regarded the body immune because its food supply was used up and the microorganisms could, therefore, no longer grow—just as yeasts cease to grow when the sugar is used up in a culture medium. It is now easy to disprove the exhaustion theory. Bacteria do not cease to grow on account of the exhaustion of the food supply, but rather on account of the production of products toxic to themselves. Further, bacteria may grow well enough in the dead tissues and fluids of immune animals, and, again, immunity may be induced by the inoculation of dead bacterial products, substances which can hardly use up food material. Recently Pasteur's exhaustion theory has been revived in a modified form by Ehrlich, who considers that there is sufficient evidence for this form of immunity in certain cases, as in cancer. Ehrlich calls it "atreptic" immunity.

Chauveau proposed the "retention" theory, the exact opposite of the exhaustion theory. This theory is also based upon the analogy of the behavior of bacterial growth *in vitro* compared to their growth within the body. It soon became evident that bacterial growth ceases even though abundant food is present, and that this inhibition is due to the retention of products of metabolism of bacterial activity. Chauveau considers that such substances are retained within the body, which thus protects it against further growth and development of the microorganisms and thus accounts for immunity.

The above theories are generalizations which have now little more than historical interest. We now know that no one mechanism of immunity will explain all cases. In some instances phagocytosis plays an important part; in others antibodies of various sorts; the side-chain theory appears to account for most of the facts in antitoxic immunity. In some cases the immunity is due to a negative property in that there is an absence of specific affinity between the poison and the cells. In others it is a positive factor and is due to the presence of substances able to neutralize the toxic action. The mechanism of immunity in some instances resides mainly in the blood and fluids; in other cases it is evidently more directly associated with cellular activity. In some instances immunity depends upon the power of immediate reaction in the sense of anaphylaxis. In all cases the mechanism is probably complex and multiple.

The unsatisfactory state of our knowledge in certain fields of immunity is well illustrated in the case of anthrax. The mechanism of protection is not at all understood in this infection, which was the first and classic illustration of a germ disease. The mechanism of immunity in common colds is also complex and obscure.

Our resistance to disease is in many cases due to a simple mechanical or chemical protection against the invasion of the pathogenic microorganisms; that is, the tissues are susceptible enough, but are guarded against the invasion of the germs of disease. Many examples may be cited in this category. Thus, one of the important functions of the skin consists in this mechanical protection of the tissues underneath. The smooth conjunctiva is protected by the constant washing of the tears and the motion of the eyelids. The lungs are safeguarded by the shape of the upper respiratory passages and the moisture of the mucous membranes, which act as a mechanical trap for many bacteria. Some of those that pass deeper are carried back by the mechanical action of the cilia. The sensitive and susceptible mucous membrane of the intestines is partly protected through the acidity of the gastric juice, which is sufficient to destroy cholera vibrios and other microorganisms susceptible to acid.

Within the body the mechanism of immunity is an adaptation of cell nutrition. The mechanism varies with different infections and in different stages of the same infection. In certain diseases the immunity seems to reside mainly in the activity of the cells. In other diseases the immunity is due chiefly to substances floating in the blood. The first is the cellular and the second the humoral theory. As we shall have occasion to see, the immune bodies in the blood are probably in all cases derived from the cells, so that the cells play the fundamental part in most cases of immunity. However, the great majority of the studies in immunology have been focused upon the changes in the blood.

This is not due to the fact that the blood alone represents these changes, but that it best represents them, and thus affords the readiest method of attacking the problem. The blood is the most fluid and most cosmopolitan of all the tissues of the body, visiting every part, bearing to each part certain substances, and removing from each part certain other substances. It is evident that it is easy to study the blood and its changes, as some of it may readily and repeatedly be withdrawn during life in order to observe its changes without in any way harming the animal. The fundamental processes of immunity within the body must all depend upon some chemical change, but we know very little concerning the chemical composition of the substances that play the chief rôle or the chemical nature of the changes. Great advances have been made in immunology despite this lack of chemical knowledge; for these advances we are indebted to experimental biology, through which we have learned the results of many effects without a knowledge of their nature or the intimate processes concerned.

Natural Immunity.—Natural immunity is an inherited character possessed in common by all individuals of a given species. It is inherent to a greater or less extent in all members of that species. It may be present at birth or develop in later years. There are very many examples of natural immunity. Thus, most of the communicable infections of man are peculiar to man; that is, the lower animals have a natural immunity to such diseases as measles, mumps, scarlet fever, typhoid fever, cholera, gonorrhea, syphilis, yellow fever, malaria, leprosy, and so on through a long repertoire.[1] Even tuberculosis, which is the most common and widespread of infections, has its own particular bacillus to which man is especially susceptible and to which the lower animals show a marked degree of natural immunity. On the other hand, man shows a high grade of natural immunity to a large number of infections to which the lower animals are subject, as rinderpest, black leg (symptomatic anthrax), Texas fever, etc.

The monopoly which man possesses of being susceptible to infections which the lower animals successfully resist is not confined to the bacteria alone, but includes many protozoa and higher animal parasites. Thus, the hookworm of man is different from the hookworm of the horse, the dog, the seal. Each host has its own species of hookworm which, though closely allied, are not interchangeable. That is, the horse has a natural immunity to the hookworm that is parasitic for man, and *vice versa*.

There is a group of infections, including the pyogenic cocci, anthrax, tetanus, malignant edema, glanders, actinomycosis, rabies, plague,

[1] It is true that some of these infections may be conveyed to monkeys or other animals by artificially introducing large amounts of the virus, but these animals do not contract these diseases naturally and therefore show a high degree of natural immunity.

foot-and-mouth disease, malta fever, tuberculosis, milk sickness, infections with the paratyphoid bacillus, ringworm, and many higher forms of animal parasites, which are common to many species in widely different genera.

There are certain remarkable facts connected with natural immunity. For example, white mice are susceptible to infections with the pneumococcus, whereas the field mouse possesses a high degree of natural resistance. When we consider how slight must be the differences in the structure, the function, the chemistry, and the metabolism in the white mouse when compared with its gray cousin, we begin to appreciate the subtle differences and perhaps complex factors upon which immunity depends. If we could find out, for example, why the goat is resistant to tuberculosis while domestic cattle are particularly susceptible, we would have the foundation for a specific preventive and cure for that disease.

Practically all the individuals of a certain species have about an equal susceptibility or an equal immunity to a given infection. These factors are more constant than commonly supposed. Laboratory animals react with certainty and with striking uniformity to an infection of known virulence, provided the virus is brought into association with certain tissues. Thus, strikingly uniform results are obtained from a given culture of plague introduced subcutaneously into the guinea pig, or of tuberculosis into the peritoneal cavity of the monkey, or of streptococci into the circulation of the rabbit, or of rabies under the dura of the dog, or of anthrax into the mouse. Man is no exception to this general statement, as far as may be judged from the data at hand. Practically all persons are alike susceptible to smallpox, yellow fever, tetanus, and many other infections. In epidemics some individuals escape. In other epidemics the disease varies greatly in severity. These apparent exceptions may not be due so much to varying degrees of immunity, but rather to variations in the dose and virulence of the virus, the channel of infection, symbiosis, and other factors.

In some cases the immunity is so weak that the balance between health and disease is quite unstable. This appears to be the case with tuberculosis in man. We possess sufficient natural immunity to tuberculosis successfully to resist small amounts of infection, but this resistance may readily be broken down by any influences which undermine our general vitality.

Natural immunity may be broken down by various means that weaken the animal, such as fasting, the production of an experimental diabetes with phloridzin, fatigue, excessive cooling of the body, as the clipping of the hair of thick-furred animals, etc. Thus, chickens are ordinarily naturally immune to anthrax, but may be infected if their

feet are kept in cold water. White rats are resistant to anthrax, but become susceptible if the hair is clipped.

Acquired Immunity.—By acquired immunity is meant a specific resistance to an infection that is not naturally inherent in all the individuals of a species, but, as the term indicates, the immunity is acquired during the lifetime of the individual. Immunity may be acquired either through some "natural" event, such as an attack of a disease, or may be "artificially" induced by the introduction of some substance, such as a serum, toxine, vaccine, or a virus.

Acquired immunity may be either active or passive. Active immunity is induced by an attack of a disease or by the introduction of a virus or suitable toxin into the system. Immunity thus acquired is active in the sense that it depends upon an aggressive stimulation of the protecting mechanism as a result of a series of reactions within the body. Passive immunity, or transferred immunity, is an antitoxic immunity. It is passive for the reason that the antibodies (antitoxin) are introduced into the body, which, therefore, takes no part in their formation. The injection of diphtheria toxine into the horse causes an active immunity in that animal; the injection of some of the antitoxin contained in the horse's serum into a child causes a passive immunity in the child. Both are acquired because horse and man have no inherent or natural immunity to diphtheria. The protection against smallpox produced by vaccination is an example of active immunity; so also is the immunity produced by bacterial vaccines.

Mixed Immunity.—Mixed immunity is a combination of the active and passive. This is used practically in plague prophylaxis and has been proposed for other infections. It consists in injecting a mixture of antitoxin serum and the appropriate bacterial virus. The advantage of this procedure consists in the fact that the active or antitoxin immunity diminishes the severe reactions which sometimes follow the introduction of a bacterial virus. It also affords an immediate protection and thereby neutralizes the negative phase which is supposed to follow an active immunization.

How Immunity May be Acquired.—Immunity may be acquired by:

(a) An attack of a disease.
(b) By the introduction of a virus.
(c) By the introduction of a vaccine.
(d) By the introduction of a chemical product (toxine).

(a) *An Attack of the Disease.*—Certain diseases, whether acquired naturally or induced artificially, leave an immunity which varies greatly in degree and duration. The following diseases leave a definite immunity of high, though variable, grade: smallpox, yellow fever, measles, whooping-cough, scarlet fever, cerebrospinal meningitis, infantile paraly-

sis, typhoid fever, typhus fever, chickenpox, mumps. Second attacks of smallpox, measles, typhoid fever, and other infections in this list are not uncommon, showing that the immunity is rarely if ever absolute.

Some diseases, such as pneumonia, erysipelas, and malaria, seem to predispose to subsequent attacks, that is, diminish resistance. Even in this class of infections there must be a certain amount of immunity, however short, else the patient would not recover.

The practice of intentionally inoculating smallpox was the first example in preventive medicine in which use was made of the fact that one attack of a disease confers immunity to a subsequent attack of the same disease. The present-day vaccination of cowpox (a modified smallpox) may be considered as belonging to this category. The principle is used to a much greater extent in veterinary practice either by using a small amount of the infection or by introducing it in an unusual way or by inoculating the animals at a time when they are found to be least susceptible. In this way a benign form of the disease is produced which protects against the severe and fatal forms. These methods are used in Texas fever, rinderpest, pleuropneumonia, anthrax, etc.

(b) *By the Introduction of a Virus Into the System.*—A distinction is made between a virus and a vaccine. If the material used contains the living active principle it should be called a virus. If the virus is dead it should be called a vaccine.[1]

The highest and most lasting degrees of immunity may be produced by the introduction of the living active principle into the system, thus imitating nature. The virus may be diminished in virulence as in anthrax, vaccinia, or rabies. A high grade of immunity to plague and cholera may be induced in man by the injection of living cultures. In the case of plague the cultures must be greatly diminished in virulence. In the case of cholera virulent strains may be used, as this disease is neither a bacteremia nor septicemia, and there is very much less danger in introducing the cholera vibrios into the subcutaneous tissue than in taking them by the mouth. This principle of introducing the virus into a resistant tissue can be taken advantage of in various infections, provided the virulence of the disease depends largely upon the channel of infection. The virulence of the virus may also be diminished by certain definite processes, such as growing the culture at an unusually high temperature, as in the case of anthrax; or by prolonged artificial cultivation, as in the classic instance of chicken cholera; or by drying, as in rabies; or by passage through animals, as in smallpox (cowpox); or by growing on unfavorable media; by the use of very small amounts of the virus, as in tuberculosis and many other infections; or by the

[1] Vaccine (*vacca*, a cow) is not a good term, but is now too deeply rooted to change.

use of closely related strains, such as the human tubercle bacillus for bovine immunization. Repeated injections of a virus induce a very high and more lasting immunity than single inoculations.

(c) *By the Introduction of a Bacterial Vaccine.*—The immunity produced by the introduction of a vaccine into the body corresponds precisely to the immunity acquired by the introduction of a virus, the only difference being that the living virus produces a more lasting and higher degree of protection than that produced by the dead vaccine. The advantages of using a vaccine instead of a virus are obvious.

Dead bacteria, when injected into the tissues, usually produce a local reaction at the site of inoculation and also a general reaction. The local reaction consists of swelling, pain, redness, and other indications of irritation and inflammation. The general reaction consists of fever, headache, pains in the muscles, especially in the back and legs, malaise, and sometimes nausea. The reactions usually come on within a few hours after the vaccine has been introduced and rarely last longer than 24 to 48 hours. It is customary to give the vaccines in the evening, for then most of the symptoms have passed by the next morning.

The vaccine is usually prepared from a fresh twenty-four-hour growth of a pure culture of the microorganism upon the surface of agar. In this way secondary metabolic products in the medium are avoided by simply removing the surface growth. When liquid cultures are used the foreign substances contained in the medium complicate the reactions. The cultures are usually killed by exposure to heat at from 53° to 60° C. for one hour. High heat, while certain to kill the virus, is undesirable, for the reason that it coagulates the albuminous substances in the germ cell and otherwise alters the chemical structure of the microorganism. The closer the vaccine approaches the virus the better the results, so far as immunity is concerned. Therefore, many investigators prefer to kill the poisons with carbolic acid, chloroform, or some other suitable germicide.

The injections are always given subcutaneously. Usually three or four injections are given at intervals of about five to ten days. Several injections produce an immunity of much higher grade and longer duration. In most instances the acquired immunity lasts from two to five years, and may be renewed.

Preventive inoculations with bacterial vaccines are now much practiced in the case of typhoid fever, plague, and cholera, and are destined to be extended to other infections. The dose and details have been discussed under each disease.

A negative phase is said to follow the introduction of a vaccine or a virus; that is, a diminished resistance appears to be produced before the curve rises. The negative phase varies in degree, depending upon the amount and virulence of the vaccine and the power of the body to react.

It varies in time from a few hours to several days. The negative phase is an assumption based upon a primary diminution in the amount of specific opsonins in the blood, but it is doubtful whether the opsonic index is a true index of the presence or absence of immunity, which is dependent upon other factors. From a practical standpoint, the negative phase can, as a rule, be disregarded; that is, bacterial vaccines are not contraindicated during the period of incubation.

Specificity.—Most of the reactions in immunology are specific—not absolutely so, but relatively; that is, antibodies, such as agglutinins, lysins, precipitins, or opsonins, usually act upon the corresponding antigen with much greater vigor than upon any other. An immunity to one disease, no matter how produced, whether natural or acquired, affords no protection against other diseases. There is, however, no absolute specificity, just as there is no absolute immunity.

Certain microorganisms and their toxic products show a remarkable predilection for certain cells or tissues. In this sense a microparasite or a toxin may be as specific in its action as a qualitative chemical reaction. Thus, there is a specific relation between tetanus toxin and nervous matter, while the poison has little or no affinity for other tissues. The poison of infantile paralysis picks out certain cells in the central nervous system upon which it acts specifically. Also in rabies the brunt of the lesions fall upon the cells of the central nervous system. The toxic products of the *Bacillus botulismus* is also a specific nerve poison, and at least one of the poisons in diphtheria toxine (toxone) acts specifically upon the nerves. The toxic substances may also react upon less important or indifferent tissues, but such action is often masked. The specific action of toxins explains in part the local immunity enjoyed by some tissues and further explains why certain viruses are comparatively harmless when introduced into the body through unaccustomed channels. We have already seen an example of this in a case of cholera when introduced into the subcutaneous tissue. In this case the subcutaneous tissue is resistant to the invasion of the cholera vibrio, and these microorganisms cannot find their way to the intestinal tract. The case of smallpox is instructive, for this is an infection for which the epithelial structures have a specific susceptibility. It is practically impossible to infect a susceptible animal with cowpox when the virus is introduced subcutaneously or directly into the circulation. The same is probably true of smallpox. When smallpox virus is introduced by inoculation upon the skin the disease is much milder than when the virus is introduced by way of the respiratory tract. Evidently the skin offers greater resistance to the smallpox virus than is offered by the mucous membranes. On the other hand, foot-and-mouth disease cannot be given to man or the cow when rubbed upon the skin, although these animals are very susceptible when this

virus is introduced into the general circulation or rubbed upon the mucous membrane of the mouth. Every worker in a bacteriological laboratory is familiar with the difference in susceptibility of different tissues and knows the importance in experimental work of bringing the virus in association with appropriate structures.

Certain microorganisms, such as tuberculosis, pus cocci, the pneumococcus, etc., have the power of affecting almost every tissue and organ of the body. No part of the body is immune to the tubercle bacillus, but even in this infection some tissues are more susceptible than others. Thus, tuberculosis of the muscle is extremely rare; the lungs and lymph nodes are especially vulnerable.

The stomach is comparatively rarely attacked by infective processes, although constantly exposed. The vaginal mucous membrane in the adult and the bladder are resistant to gonorrheal inflammations. There are many similar instances of specific immunity of tissues.

The specific action of toxins gives us a ready reason why certain species of animals are immune to certain infections. In this case the immunity is not the result of any special or specific reaction, nor is it the result of any positive character possessed or acquired by the body, but is a negative trait entirely, due to the absence of specific chemical affinity between the cells and the toxin. The turtle is immune to tetanus because there is no combining affinity between the nerve cells of the turtle and tetanus toxin. The immunity, therefore, depends upon the absence of the appropriate cell receptors. Rats are highly immune to diphtheria toxin and hogs to snake venom. In these cases antitoxin cannot be demonstrated in the blood of the rat or the hog, and, so far as can be determined, when the toxin is injected into these animals it is not neutralized in the body. The simplest conception of the mechanism of immunity in these cases is to regard it as depending upon a negative factor resulting upon the absence of suitable receptors in the sense of Ehrlich's side-chain theory.

Local and General Immunity.—Local and general immunity depends upon this variation in susceptibility of the different tissues to different infections. It is doubtful if there is a true general immunity in any case, for a general immunity is in almost all instances based upon a local resistance. Even antitoxic immunity in diphtheria, due to the antibodies in the general circulating blood, is the result of a localized neutralization in which many of the organs and tissues of the body take no part. There are many examples of local immunity. *Trichina spiralis* affects especially the muscles and never the bones. Diphtheria seldom extends down the esophagus. The most marked example, perhaps, is the almost perfect local immunity of the scalp to ringworm in adults, which contrasts so markedly with the absolute susceptibility of children, whereas the susceptibility of the skin

of the body to the same parasite is, if anything, greater in adults than in children (Emery).

Many remarkable instances of local immunity are shown by the tissues and must be familiar to all. Thus, erysipelas does not, as a rule, extend into the subcutaneous tissues, although the streptococcus may be there; rarely does it extend back into the area of the skin recently affected.

The immunity of a part is increased or diminished by the presence or absence of an adequate blood supply. As a rule, very vascular structures enjoy a comparative immunity to infections which frequently attack other tissues relatively poor in blood supply. It may be stated as a general rule that the more copious the supply of healthy circulating blood the greater the resistance to infection, and *vice versa*. This largely accounts for the local immunity enjoyed by the mucous membrane of the mouth and lips, which are constantly exposed to wound infections. Herein we also have an explanation of the utility of fomentations and other hot applications in the initial stages of an infective lesion. The same explanation is applied to Bier's method of passive congestion, in which an excess of blood (though partly stagnant) is made to flush the tissues. The local immunity of the part may be diminished by a local anemia from any cause, by the presence of dead or injured tissue, by the action of irritants, trauma, etc.

Metchnikoff has pointed out that in many infections general protection is in inverse ratio to the local reaction at the site of introduction of the virus. A severe and prompt local inflammatory reaction indicates an active power of protection. The increased volume of blood, the cells, the fluids of the blood and tissues are concentrated about the invading bacteria to wall them off and destroy them, that is the immunity of the body against a general infection frequently depends upon the promptness and the activity of the local power of reaction.

Some infections, notably streptococci, plague, or organisms belonging to the hemorrhagic-septicemic group, may invade the body with little or no local inflammatory reaction; that is, little or no barrier is set up against these microorganisms, they invade the blood and tissues without resistance and thus cause fatal septicemias.

Bacillus Carriers or Immunitas Non Sterilans.—Upon recovery from an infective process the body usually rids itself completely of the infecting agent. In other words, the immunity which follows an attack of an infectious disease is usually associated with a power the body has of disinfecting itself. In most cases the patient is convalescent or completely restored to health before the cause of the disease has disappeared from the tissues. This bespeaks a vigorous protecting mechanism, but when this resistance is lowered for any reason a relapse may ensue.

In many instances recovery takes place, but the living virulent microorganisms continue to live in the body. This constitutes immunity without sterilization, a term introduced by Ehrlich, though a more precise expression would be "immunity without disinfection." Such persons are now known as "bacillus carriers." The immunity protects the carrier but endangers his fellowmen. Bacillus carrying is common in diphtheria, typhoid fever, cholera, pneumonia, epidemic cerebrospinal meningitis, influenza, and many other bacterial infections. Protozoon carriers are also a common phenomenon. The best examples are found in malaria, trypanosomiasis, Texas fever in cattle, etc. Analogous instances are also found in the higher parasitic worms in which the individual who carries the parasite is not affected. Thus, the negro and the Filipino show a relatively high degree of immunity to the hookworm and thus endanger their more susceptible white companion.

An *acute* bacillus carrier is one who sheds the specific agent of the disease for a few weeks—four to six following convalescence. A *chronic* bacillus carrier is one who harbors and discharges the specific agent a longer period than six weeks. A *temporary* carrier is one who harbors the specific infective agent, although he himself has never had symptoms of the disease. Temporary carriers may be acute or chronic, depending upon the length of time they harbor the particular parasite.

Bacillus carriers play an important rôle in spreading infections. They explain many mysterious facts in the epidemiology of diphtheria, typhoid fever, cholera, cerebrospinal meningitis, malaria, etc. The bacillus carrier is sometimes a danger to himself. This is seen in diphtheria, pneumonia, influenza, and sometimes in typhoid and cholera. Thus, a person may carry the pneumococcus in his throat for years awaiting certain favorable conditions for infection before he contracts the disease. The same is more or less true of other carriers.

While it is undoubtedly true that bacillus carriers play a very important rôle in spreading infection from man to man, the relative importance compared with other modes of transmission cannot be stated in percentage. The subject is still too young for definite quantitative figures. There is no doubt that bacillus carriers are more important in some diseases than others and play a variable rôle under different circumstances in the same disease. In our studies of typhoid fever in Washington one carrier was discovered in the examination of 986 healthy individuals. This would mean approximately 300 typhoid bacillus carriers in the District of Columbia. If this proportion is correct, it would account for the endemicity of typhoid fever in Washington. Perhaps the residual typhoid fever in many places is largely kept alive through bacillus carrying, and there is little doubt that the gradual decline of typhoid fever after great sanitary reforms, such as the

change from polluted to pure water, is due to the decrease in the number of carriers. It now seems evident that polluted water and infected milk will not always cause the disease directly in the persons drinking these fluids, but may produce numerous carriers who either contract the disease themselves subsequently or give it to others by passing the virus on in a more concentrated and virulent form, or to more susceptible individuals.

It is evident from the nature of the case that the cure and control of bacillus carriers is one of the vital problems in preventive medicine. It is not only largely through them that infection is spread, but the infections themselves are kept alive in these carriers, who bridge over the interval between outbreaks. It is quite conceivable that with our modern methods of isolation and disinfection certain diseases would soon cease to exist were it not for *immunitas non sterilans.*

Immunity is, therefore, a double-edged sword, in that it protects the carrier but endangers his neighbor. The control of bacillus carriers is a difficult problem. Such unfortunate persons cannot always be imprisoned, nor is strict isolation always necessary. It is sufficient in the case of typhoid fever to restrict the activity of the carrier. Thus, a typhoid carrier should not cook, prepare, or handle food, or have anything to do with the production or distribution of milk. We have no satisfactory cure for carriers; this is a problem for the future; but their number may be lessened—this is a problem for the present.

It should always be remembered that the number of carriers will diminish proportionately with the number of cases of any infection, and that every improvement in the water supply, the milk supply, the food supply, and our sanitary conditions generally will have a tendency to sharply diminish the number of carriers in any given infection. Therefore, while isolation, disinfection, and other methods used to control the spread of infection will never be completely successful as long as the carrier is omitted, nevertheless, these methods are entirely justified even though only partially useful. It is the duty of public health officers to check the spread of infection wherever it may be found. In time ready methods of recognizing bacillus carriers and means of neutralizing their potential danger will be more effective than is now possible.

Latency is closely allied to bacillus carrying. The malarial parasite may remain latent in the spleen and other internal organs for years, during which time the person remains in good health. But when the resistance is reduced by exposure, fatigue, starvation, or other depressing influences the disease again breaks out. The gonococcus may also remain latent for years. I am familiar with one instance in which the tubercle bacillus remained latent in the axillary glands for 10 years and then became active owing to a condition of depressed vitality. Typhoid ostitis may develop years after an attack of typhoid

fever, and we can only assume that the bacilli have remained latent in the tissues all that time. The phenomenon of latency also occurs in rabies, tetanus, and other infections.

Lowered Resistance.—The factors which lower our general resistance to disease are many and varied. The condition known as depressed vitality, lowered tone, general debility, weakened constitution, and terms of similar import imply a condition in which immunity is lowered in a general and not in a specific sense. The principal causes which diminish resistance to infection are: wet and cold, fatigue, insufficient or unsuitable food, vitiated atmosphere, insufficient sleep and rest, worry, and excesses of all kinds. The mechanism by which these varying conditions lower our immunity must receive our attention, for they are of the greatest importance in preventive medicine. It is a matter of common observation that exposure to wet and cold or sudden changes of temperature, overwork, worry, stale air, poor food, etc., make us more liable to contract certain diseases. The tuberculosis propaganda that has been spread broadcast with such energy and good effect has taught the value of fresh air and sunshine, good food, and rest in increasing our resistance to this infection.

There is, however, a wrong impression abroad that, because a lowering of the general vitality favors certain diseases, such as tuberculosis, common colds, pneumonia, septic and other infections, it plays a similar rôle in all the communicable diseases. Many infections, such as smallpox, measles, yellow fever, tetanus, whooping-cough, typhoid fever, cholera, plague, scarlet fever, and other diseases, have no particular relation whatever to bodily vigor. These diseases often strike down the young and vigorous in the prime of life. The most robust will succumb quickly to tuberculosis if he receives a sufficient dose of the virulent microorganisms. A good physical condition does not always temper the virulence of the disease; on the contrary, many infections run a particularly severe course in strong and healthy subjects, and, contrariwise, may be mild and benign in the feeble. Physical weakness, therefore, is not necessarily synonymous with increased susceptibility to all infections, although true for some of them. In other words, "general debility" lowers resistance in a specific, rather than in a general, sense.

The mechanism by which the various causes that lower vitality and increase susceptibility act is in most cases quite obscure. Here is a field for laboratory research in immunology that offers rich reward of immeasurable practical good. Some of the factors concerned will be briefly discussed.

Exposure to wet and cold, especially in combination, is a frequent source of lowered resistance. The exact way in which such exposure acts is not definitely known, but laboratory researches offer material

for a number of suggestions. Emery[1] sums up our knowledge upon this subject as follows:

"Immunity is to a very large extent a function of the leukocytes, which are specialized cells to which the defense of the body is entrusted. Now, the functions (movement and phagocytosis) which can be easily investigated are found to be dependent in a very high degree on temperature, acting best at the temperature of the body, or slightly above; and it is highly probable that the more subtle functions of the leukocytes may be similarly depressed by a low temperature. The exposure of the skin to cold, especially if the animal heat be abstracted more quickly by evaporation of moisture on the surface, will lead to a cooling of the blood which circulates through it, and hence to a slight, though appreciable, cooling of the whole blood. This, it is true, is soon compensated for, and no great amount of cooling of the whole body occurs; but, even so, it is quite possible that the periodical chilling of the leukocytes during their repeated passages through the cold skin may be sufficient to diminish greatly their functional activity, and to lower the resistance to a point at which infection may occur, and when once pathogenic bacteria have gained a foothold the resistance will for a time tend to decrease. There is also some evidence going to show that exposure to cold may lessen the production of the defensive substances which occur in the blood (alexin, antibodies, etc.), though this is not fully proved. It is worthy of note that the loss of immunity due to the action of cold and wet on one part of the body (such as the feet) is a *general* one, and may result in a nasal catarrh, an attack of pneumonia, acute rheumatism, etc., according to the nature of the infection at hand. It is not necessarily a local infection of the chilled region. This is very well shown experimentally. Fowls are immune to anthrax, but are rendered susceptible if they are kept for some time standing in cold water; and this acquired susceptibility is then a general one, and not merely of the feet.

"Cold and wet, as is well known, have less action when accompanied by energetic muscular exercise, so long as this does not reach the extent of undue fatigue. This is not because less heat is lost during exercise. The reverse is the case. The suggested explanation is that the muscular metabolism leads to an increased production of heat, and at the same time the cutaneous capillaries are dilated and the heart accelerated, or that the circulation of blood through the skin occurs quickly; further, the internal temperature of the body may actually be raised several degrees. The result is that the temperature of any given leukocyte never falls much below normal, if at all, since it comes from the internal regions where the temperature is raised, passes rapidly through the skin, and returns again to the interior of the body.

[1] "Immunity and Specific Therapy," 1909, p. 9.

"The effect of *fatigue*, either alone or in conjunction with cold and wet, is also well known, and is one reason for the excessive mortality from disease of armies in the field. It is less explicable, but may probably be connected in some way with the presence in the blood of katabolic products of muscular activity, which have an injurious action on the cells of the tissues in general and on the leukocytes in particular. Further, the metabolic products formed during the action of the muscles are acid in reaction, and it is found that some at least of the protective substances which occur in the blood (alexins and opsonins) act best in alkaline medium. This diminution of immunity after muscular fatigue is manifested in animals as well as in man. White rats which have been made to work in a revolving cage are more susceptible to anthrax than normal white rats, the preëxisting immunity being broken down."

De Sandro [1] "injected dogs, rabbits, guinea pigs with typhoid toxins after severe muscular strain. Under the influence of the chemical changes induced by the physical strain, the nervous exhaustion, fatigue of the heart, and disturbances in the blood production, the defensive powers were evidently much weakened; phagocytosis was reduced and also the chemotactic power of the cells, the bacteriolysins, antitoxins, agglutinins, and opsonins showed a marked falling off."

Insufficient and unsuitable food is a prime factor in undermining vitality and lowering resistance. The influence upon health of food poor in quality or lacking in quantity is a matter of common experience, but the scientific explanation of the way in which this result is brought about is not at all clear. First of all, it must be remembered that starvation or improper food does not depress immunity to all infections, but lowers resistance only to certain infections. It was formerly supposed that famine was the direct cause of pestilence. In fact, in India it has commonly been stated that "plague follows famine with some regularity," but we know now that plague in man is secondary to the disease in rats and is transmitted through the flea. Relapsing fever was formerly called famine fever, and outbreaks of typhus fever were frequently connected with famine, but we know now that the former is transmitted by the tick and the latter by the louse. It is evident that famine may be indirectly a cause of epidemic outbursts without necessarily depressing immunity. Famine is usually accompanied by misery and squalor and an increase of vermin and other factors that favor the transmission of disease.

Tuberculosis, of all diseases, is favored by insufficient and unsuitable food. This is an infection in which poor nourishment lowers, and good nourishment raises, our immunity. Poor and insufficient food, however, is usually associated with poverty, insufficient clothing, un-

[1] *Riforma Medica*, Naples, Aug. 1 & 8, Nos. 31 & 32.

cleanly habits, vitiated atmosphere, overwork, insufficient rest, and other depressing influences, so that it is difficult to assign relative importance to any one of these factors. For this reason we may perhaps be led to exaggerate its importance; and, while it is, of course, true that semistarvation, in common with other weakening influences, does pave the way for infective processes, we do not find that a supply of food restricted enough to cause a marked reduction of the bodily strength and some degree of anemia is necessarily associated with any infective disease, though the patient may live under conditions in which infective material is present in abundance. This is well seen in fasting men, in hysterical anorexia, and in patients with impermeable esophageal strictures. The blood, it may be pointed out, is not one of the tissues that suffers first in starvation, and its importance to the body in many ways is so great that it is kept in good functional activity while other tissues waste quickly.

There is a general belief that exposure to infection is less dangerous after a meal than upon an empty stomach. There is little ground for this belief, unless we take into consideration the notable increase in the number of leukocytes in the peripheral blood during active digestion. It was recognized long ago that wounds inflicted during autopsies are much more dangerous when received while fasting than during the process of digestion, and it is possible that this may be due to some extent to the increased number of leukocytes which occur in the blood during the process. Further, infection reaching an empty stomach has greater chances of passing into the small intestines than if it reaches the stomach after a full meal when acidity, time, and the digestive enzymes have a chance to destroy the microorganisms. This may be of importance in cholera, typhoid, dysentery, and other intestinal infections.

Exposure to a vitiated atmosphere, if of long duration, is one of the potent causes of breaking down resistance. Here again, however, immunity is lowered in a specific and not in a general sense. Thus, vitiated air renders the individual more susceptible to tuberculosis, pneumonia, common colds, and other acute respiratory affections. On the other hand, it can have little influence in determining the infection of most of the communicable diseases, although the lowered tone of the body caused by vitiated air may influence the severity of the attack. The mechanism by which vitiated air increases susceptibility is not understood at all. The subject is discussed in the chapter upon air.

Excesses of all kinds, symbolized by Bacchus, Venus, and Vulcan, are mighty factors in lowering vitality and in increasing susceptibility to certain diseases. In this category are also found worry, overwork, loss of sleep, and fatigue.

Certain *drugs,* of which the most important is alcohol, have an im-

portant action in lowering resistance. Emery states that: "The liability of alcoholic subjects to pneumonia and some other infective diseases is well known, and in them the prognosis is more than usually unfavorable. We have but little knowledge of the action of alcohol in this respect. It may be that it acts as a direct inhibitant of the activity of the leukocytes, and it is known to destroy certain delicate defensive substances (alexins and opsonins) which play some part in the defense of the body against microbic invasion, but it is not known whether these effects are actually manifested in the circulating blood. It is also possible that alcohol tends to inhibit the formation of these defensive substances.

"Alcohol tends to lower the temperature of the body by increasing the amount of heat lost. It dilates the superficial vessels and accelerates the heart action in a way somewhat similar to muscular exercise, but does not, like it, raise the temperature of the interior of the body. Hence the effect of alcohol in conjunction with cold and wet is to increase their ill effects. More blood is forced through the chilled skin and more heat is lost. The injurious effect of alcohol during exposure to cold is well known. The results, however, are different when alcohol is taken after exposure, and when the sufferer has reached warmth and shelter. There the increased flow in the cutaneous capillaries leads to a warming of the skin and consequent cessation of the chilling of the blood, although the loss of heat may go on."

Ehrlich's Side-chain Theory of Immunity.—Ehrlich's [1] side-chain theory is a brilliant chemical conception, giving the only satisfactory explanation we have of some of the phenomena concerned in immunity. In one sense it has been likened to Weigert's teachings of inflammation and the process of repair in so far that cognizance is taken of nature's prodigality. For instance, a much larger amount of material is thrown out than necessary to repair a wound. So, too, in antitoxic immunity a much larger amount of antitoxin is produced than necessary to neutralize the toxin.

In Ehrlich's conception the fundamental processes of immunity reside in the cells of the body. These cells are attacked by the poison, and if not destroyed are stimulated to an overproduction of "antibodies" capable of combining with and neutralizing the poison.

Just what cells of the body play the most important rôle in the production of this form of immunity is not exactly clear. It may be, as Ehrlich supposes, that this power resides in any organ or tissue.

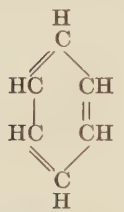
According to Ehrlich, the hungry protoplasm of any cell, with its

[1] Ehrlich: "Die Wertbemessung des Diphtherieheilserums und deren theoretische Grundlagen." *Klin. Jahrb.*, Jena, VI (2), 1897, pp. 299-326.
——"Ueber die Constitution des Diphtheriegiftes." *Deut. med. Woch.*, Leipzig, XXIV (38), 1898, pp. 597-600.
——Croonian lecture. "On immunity with special reference to cell life." *Proc. Roy. Soc.*, London, LXVI, pp. 424-448, pls. 6-7.

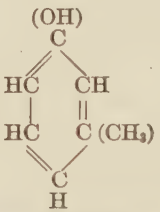
complicated molecule, having side chains of various combining affinities ready to unite with suitable food molecules brought to it by the blood and body juices, lies at the foundation of his explanation of the chemical production of the antitoxin. It is strange that the same combining affinity should exist between the protoplasm of the cell and the proteid molecules that furnish it food, as between the cell protoplasm and the toxins of the bacterial poisons.

In considering Ehrlich's[1] side-chain theory it is necessary to disregard the microscopic structure of the cell and to think of the protoplasm as consisting of living molecules of extraordinary chemical complexity. The molecule of protoplasm has a central "nucleus" with "side chains," "lateral chains," or "bonds" of varying combining capacities. These "side chains" serve to bind the molecule to other molecules having proper combining affinities.

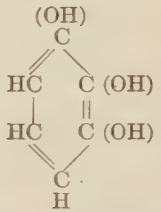
This arrangement of molecules with side chains is a well-known occurrence in organic compounds. The bezol ring forms one of the best and simplest examples.



Benzol C_6H_6



Metacresol $C_6H_4(CH_3)$ (OH)



Pyrogallic acid $C_6H_3(OH)_3$

By replacing one of the H atoms in the bezol ring with the methyl radical (CH_3) we have toluol; by replacing one of the H atoms with the hydroxyl group (OH) we have phenol; by substituting two hydroxyl groups we have resorcin; three, pyrogallic acid, etc.; by substituting one hydrogen atom of the ring with the hydroxyl radical and another one with the methyl radical we have the cresols.

These simple illustrations from well-known organic compounds illustrate the central molecular mass with its side chains and combining affinities, to which the molecule of protoplasm is likened.

In applying this analogy to the molecule of protoplasm the name "receptor" is given these side chains, or secondary atomic complexes of the molecular group. Contrary to the simple analogies above given, each molecule of protoplasm has many different kinds of receptors, as shown by the schematic diagram in Fig. 44. These receptors have a specific affinity for the molecules of food, and also combine with the toxic molecules.

The *toxin* molecule, according to Ehrlich, consists of two important

[1] Ehrlich: "Die Wertbemessung des Diphtherieheilserums und deren theoretische rundlagen," *Klin. Jahrb.*, Jena, VI (2), 1897, pp. 299-326.

parts. One is known as the *toxophore* group, the other as the *haptophore* group.

The *toxophore group* of the toxin is that portion of the molecule which exerts a poisonous effect upon the protoplasm of the cell. This group is less stable than the haptophore group.

Fig. 48.—The Cell with Its Various Combining Groups or Side Chains, Known as Receptors. Various toxins are shown having specific affinity for the proper shaped receptors.

The *haptophore group* is the seizing or combining portion of the *toxin* molecule ($\alpha\pi\gamma\omega$, to seize or attack). The haptophore group of the *toxins* have specific combining affinities for the receptors of certain cells, which in part explains the selective action of these poisons.

Toxines such as diphtheria toxine gradually diminish in toxicity, but retain the same power of chemical combination with the antitoxin. This phenomenon explains the formation of *toxoids*.

Ehrlich inferred the presence of the *toxoid* from the following simple experiment: He had a toxine which required 0.003 c. c. to kill a guinea pig. After nine months this poison weakened, so that it required three times as much, that is, 0.009 c. c., to kill a guinea pig. Nevertheless, the combining power of the toxine for antitoxin remained the same.

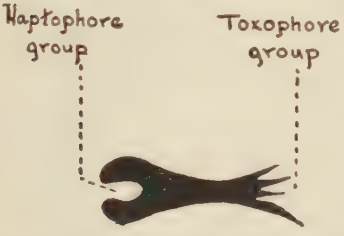


Fig. 49.—The Toxin Molecule; Showing the Haptophore (Combining) Group, and the Toxophore (Poison) Group.

Toxoids are altered *toxins*. They consist of the toxic molecule in which the toxophore group has been destroyed, leaving only the haptophore or combining group, which, while able to satisfy the combining affinities of the antitoxin, is no longer able to poison the protoplasm of the cell.

The diphtheria bacillus, during the process of its growth and multiplication in the body or in an artificial culture medium, produces several poisons, one of which is known as diphtheria *toxin*. As above stated, the diphtheria *toxin* consists of a toxophore and haptophore group. In the body the latter unites chemically with the receptors of the cells. When this takes place one of two consequences may result: either (1) the cell is so severely poisoned that it dies, or (2) the living molecule of protoplasm is stimulated so as to excite a defensive action by the reproduction of the receptors. Continued stimulation produced by the periodical injection of toxine results in an overproduction of receptors, which finally

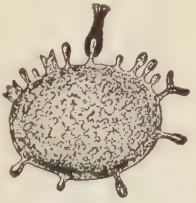


Fig. 50.—The First Stage of Antitoxin Formation: a Toxin Molecule Anchored to a Receptor.

loosen and float free in the blood serum and body juices. Receptors fixed upon the cells are called sessile, and those that leave the cell are spoken of as free receptors.

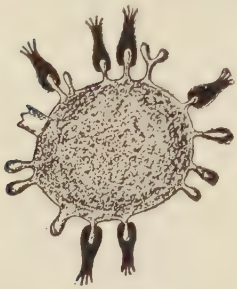


Fig. 51.—The Second Stage: Continued Stimulation Causes a Reproduction of Receptors.

Antitoxin consists of these free receptors floating in the blood serum. If we now introduce *toxin* into the blood, it is immediately neutralized by combining with the free receptors through its haptophore group. All the combining affinities of the *toxin* are thus satisfied or saturated, so that the *toxin* is no longer able to unite with the receptors still attached to the cell, and the poison is thus rendered harmless.

It is by no means a necessary corollary of the side-chain theory, as is often supposed, that the receptors are found only in those organs upon which the poisonous effects of a toxin are particularly manifested. On the contrary, Ehrlich and Morgenroth [1] believe that receptors capable of combining with the toxin are produced in many different parts of the body, especially in tissues and organs having the power of anchoring the toxin without causing serious poisonous effects.

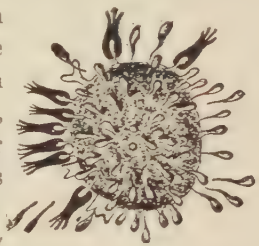


Fig. 52.—Third Stage: the Receptors Beginning to Leave the Cell.

The connective tissue is believed to be specially rich in receptors, evidenced by the local reaction caused by the subcutaneous inoculation of diphtheria toxine, ricin, abrin, and similar poisons. In fact, one would not be far wrong in assigning a particular significance, in the production of receptors, to just those organs which show unimportant vital response, because in such tissues the injurious effects of the toxophore group are absent or of such diminished importance that the regenerative powers of such tissues are not retarded.

Fig. 53.—Fourth Stage: the Receptors Have Left the Cell and Float Free in the Blood—Antitoxin.

The presence or absence of receptors capable of binding the toxine, as well as their number and distribution, are factors which determine the susceptibility of different species of animals against the various toxines. These factors also determine the individual variations in the susceptibility to poisons and further explain some instances of natural immunity to toxines.

[1] Ehrlich, P., & Morgenroth, J.: Wirkung und Entstehung der aktiven Stoffe im Serum nach der Seitenkettentheorie. Handbuch der pathogen Mikroorganismen, W. Kolle, and A. Wassermann, Jena, 1904.

An example is given by Sachs,[1] who studied the reaction of guinea-pig blood against arachnolysin, a toxine found in spiders. In this case the complete immunity of the red blood cells of the guinea pig against arachnolysin is accounted for by the entire absence of the proper receptors, while the susceptibility of the red blood cells of the rabbit to very small quantities of this poison is accounted for by the strong combining affinity which exists between these cells of the rabbit and the arachnolysin.

Fig. 54.—The Neutralization of a Toxin by Antitoxin; the Free Receptors in the Blood Have United with the Toxin = Antitoxic Immunity.

In some cases the production of receptors may apparently be traced in the development of certain species. Cannus and Gley[2] have followed the development (?) of the receptors in the red blood cell of the rabbit toward the hemolysin found in eel serum. Young rabbits are much less susceptible to this poison than adult rabbits, which is accounted for by Ehrlich as being due to a gradual development of the receptors having proper combining affinities for the hemolysin found in the eel serum.

Fig. 55.—The Second Order of Immunity, Showing Complement and Immune Body.

The union between the receptor of the cell and its poison is not always a direct one, as described above, but sometimes takes place through the intervention of a second body, known variously as the amboceptor, *zwischenkörper,* immune body, sensitizer, fixative, preparative, desmon, etc.

This second order of immunity is particularly evident in the poisons that have a lytic or dissolving action upon bacteria or the cells of the body, such as the bacteriolysins, hemolysins, and other cytolysins. The poisonous bodies in this order of immunity are usually spoken of as "complement," but also as the "alexin" (Buchner) or "cytase" (Metchnikoff).

One of the remarkable facts connected with the phenomena of the lytic poisons is that the poison itself (the complement) is normally present in the blood. Complement is thermolabile, that is, it has less resistance to heat than the intermediary body, which is thermostabile. According to Ehrlich's theory, immunity can only be obtained against the intermediary body, which is believed to be specific.

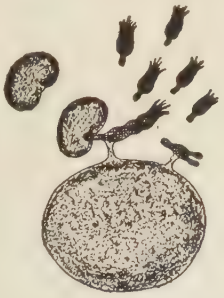


Fig. 56.—The Third Order of Immunity, Showing an Immune Body Having Two Affinities.

[1] Sachs, Hans: "Hofmeisters Beitr.," Bd. 2, h. 1-3.
[2] Quoted by Ehrlich, loc. cit.

Ehrlich compares the intermediary body with diazobenzaldehyde, which by means of its diazo group is capable of combining with a series of bodies, such as aromatic amins, phenols, ketomethyl bodies, etc., while by means of its aldehyde group it may combine with a different series, such as the hydrazins, ammonia radicals, and hydrocyanic acid. Phenol and hydrocyanic acid will not directly combine, but, with diazobenzaldehyde acting as an intermediary body, these two substances can be brought into combination. Pushing this comparison further, we may say that the aromatic body, or the phenol, represents a constituent of the blood corpuscle. The diazobenzaldehyde is the intermediary body, while the poisonous hydrocyanic acid constitutes the complement.[1]

Welch[2] very ingeniously extended Ehrlich's conception of immunity to the bacterial cell. According to Welch's views, the bacterial cell has the same power of defensive action against the poisons produced by the cells of higher animals that they have against the toxic products of the bacteria.

In other words, there is a chemical battle. Both the bacterial cell and the body cell excrete poisonous substances against each other, and both in turn are building up a chemical defense against the action of these respective poisons.

Antitoxic Immunity.—In order to understand antitoxic immunity it is necessary to consider the nature and action of toxins, the formation and production of antitoxins, and the reaction between toxins and antitoxins and related subjects.

TOXINES

Bacteria produce many different kinds of poisonous substances, but not all of these are toxines in the specific sense in which that term is now used. A toxine may be defined as a specific poison elaborated by bacterial metabolism; it is soluble in water; poisonous in minute amounts; reproduces the essential symptoms and lesions of the disease; acts only after a period of incubation; and produces antibodies, especially antitoxin. The toxines are thermolabile, unstable, and have a complex chemical structure.

Toxines are known only by their effects upon animals; they cannot be recognized in any other way. Presumably they belong to the higher proteins, but nothing definite can be stated concerning their chemical structure. They have never been isolated in pure form; they are not toxalbumins, as was once believed, and they only have a remote analogy

[1] Vaughan and Novy: "Cellular Toxins," 1902, p. 131.

[2] Welch, William H.: "Huxley lecture on recent studies of immunity with special reference for their bearing on pathology." *Bull. Johns Hopkins Hosp.*, Balto., XIII (141) Dec., 1902, pp. 285-299.

to the enzymes. Toxines may be globulins, at least they come down in the globulin fraction. They may readily be precipitated with ammonium sulphate, for example, but whether they are mechanically carried down in the precipitate is not known. The toxine molecule is at least small enough to readily pass through the pores of the finest porcelain filter, and large enough not to dialyze through a membrane.

There are three well-known toxines: diphtheria, tetanus, and botulismus. A number of bacteria, such as cholera, dysentery, pyocyaneus, and others, produce a certain amount of toxic substances soluble in water, but it is very doubtful whether they are true toxines in accordance with the above definition. Bacteria produce many poisonous substances other than the true toxins, such as acids, alkalies, nitrites, ptomains, alcohol, hydrogen sulphid, etc. Some of these substances may play a part in the pathogenesis of disease, but while they are poisonous they are not true toxines.

Toxines are sometimes divided into exotoxins and endotoxins. The former are the true or soluble toxines; the latter are insoluble under ordinary circumstances, and differ markedly from the true exotoxins. The endotoxins will be considered separately.

The tubercle bacillus, the bacillus of glanders, and other microorganisms produce soluble toxic substances specific in nature but quite different from the true toxines, in that they are harmless to a normal animal, but poisonous to one suffering with the specific disease. Tuberculin, mallein, and similar "toxins" are very stable, resist heat and other influences, do not produce the specific lesions and symptoms of the disease, do not stimulate antitoxin formation, and in other ways differ from the genuine toxines.

A toxine is produced as a result of bacterial metabolism within the body, but whether it is a secretion, an excretion, or a product of the action of the bacteria upon the medium (as alcohol and carbon dioxid are produced by yeasts) is not known. It is known, however, that toxines do not result simply from the breaking down of the dead bacterial cells, as was once stated.

It is now evident that different groups of bacteria produce poisons that differ essentially in chemical structure as well as in physiological action, just as different species of higher plants produce various poisons that differ markedly in composition and physiologic action.

Very few of the bacterial poisons are injurious when taken by the mouth. Diphtheria and tetanus toxines are practically inert, being destroyed largely by the digestive juices and not being absorbed in any harmful amount. Enormous doses of these toxins may be administered by the mouth to susceptible animals without appreciable harm. The antibodies and the consequent slight immunity probably produced by the absorption of dead bacterial cells and their toxic products (such as

typhoid and tubercle) from the digestive tube is an entirely different phenomenon and does not apply so far as the true toxins are concerned. There is one notable exception in the case of the toxine of the *Bacillus botulismus,* for this poison is absorbed by the intestinal mucosa, and it is in this way that it produces its harmful effects in man.

There are several poisons produced by higher plants that resemble the true bacterial toxines in all important respects. Among them are: *ricin* from the castor bean, and *abrin* from the jequirity bean. These toxines of vegetable origin are known as phytotoxins. They are soluble, act only after a period of incubation, are exceedingly poisonous in small amounts, are destroyed by heating, and produce specific antibodies. They are probably of protein nature, according to Osborne, Mendel, and Harris, who obtained ricin in very pure form. These poisonous substances of vegetable origin have more than theoretical interest, for it was through a study of their action that Ehrlich first obtained a deeper insight into the nature of toxines and antitoxic immunity.

There are poisons in the animal kingdom which closely resemble the toxines, such as the venom of snakes, scorpions, spiders, wasps, etc.

True toxines are unstable and are readily affected by heat, sunlight, acids, and various chemicals. They are much more unstable in solution than in dry powdered form. Tetanus toxine is more labile than diphtheria toxin, but when precipitated with ammonium sulphate and preserved as a dry powder in a vacuum tube, and in a cool, dark place it may be kept without deterioration for several years. Diphtheria toxine, in solution, weakens rapidly at first, and then comes to a stage of equilibrium which it maintains indefinitely if preserved in a cold, dark place and protected from the oxygen of the air.

The poisonous properties of toxines of diphtheria, tetanus, and botulismus are destroyed at once by boiling, and at 65° C. in a short time. At 60° C. for one hour they lose most or all toxic power.

It has been stated that one of the characteristics of the toxines is that they are poisonous in exceedingly small amounts. Thus, .000,000,05 of a gram of a partially purified tetanus toxine will kill a mouse. Diphtheria toxines have been obtained so that .0008 c. c. of the unconcentrated fluid (crude filtrate) will kill a guinea pig.

A true toxine reproduces the essential symptoms and essential lesions of the disease. In this sense they have a specific action. The symptoms produced in a susceptible animal by the inoculation of tetanus toxine cannot be distinguished from the disease naturally contracted. The symptoms produced by the injection of diphtheria toxine closely resemble diphtheria, including coagulation necrosis at the site of the injection, fever, depression, post-diphtheritic paralysis, etc. The symptoms following the ingestion of the toxine of the *Bacillus botulis-*

mus are an exact counterpart of the disease produced by eating food containing the poison of this microorganism. This specific action is very important, and, if it were more generally known, would save many mistakes in experimental biology and its application to serum therapy. It is comparatively easy to obtain useful antitoxins from true toxines. On the other hand, it seems to be impossible to obtain antitoxines of any therapeutic potency from other bacterial poisons. Thus, tuberculin and mallein and other "toxins" do not stimulate antitoxic production and the so-called antitoxic sera thus produced have no protective or curative value. It must not be forgotten that only a comparatively few infections depend upon toxines and may be prevented or cured by corresponding antitoxins.

One of the characteristics of the true toxins is that they act only after a period of incubation. In this respect they resemble the natural disease. Simple chemical poisons may act at once, but the toxines produce no apparent effect until a definite time elapses after they have been introduced into the system—even when overpowering doses are administered. Thus, the ordinary period of incubation when tetanus or diphtheria toxine is injected into a susceptible animal is several days. When enormous amounts are injected this may be reduced to about 8 or 12 hours, but never less. The period of incubation is inversely proportional to the amount of poison injected. The longer the period of incubation the milder the symptoms; when the period of incubation is short the result is almost invariably fatal. The cause of the period of incubation is not well understood. A certain length of time is required for the toxine to reach the susceptible cells. This varies especially in the case of tetanus, which travels up the nerves. After the poison reaches the cells further time is required to combine chemically, and then more time to produce the injury. On account of the period of incubation large amounts of toxine may be present in the circulating blood before the appearance of symptoms. Thus, in horses enough tetanus toxin has been found in the blood two days before the onset of symptoms to kill a guinea pig, when only 0.1 c. c. of the blood serum was injected.

The distribution of the toxines in the body is unequal. Most of the poison unites with the cells; some is destroyed and some neutralized if antitoxin is present. Most of it probably unites with the cells, as it soon disappears from the blood. Tetanus toxine may remain a long time in the blood of an insusceptible animal. Thus, Metchnikoff could demonstrate the presence of tetanus toxine in the tortoise four months after the injection. After tetanus toxine is injected it soon disappears from the blood, but if the tissues are injected into a susceptible animal tetanus is produced, for it is now known that this poison has a specific affinity for the motor nerve endings. In the case of fowls it

seems that this power of combining with the tetanus toxine is most marked in the leukocytes. Toxines will not combine with all cells indifferently. They have a specific combining affinity for certain cells. Tetanus toxine has a special affinity for the cells of the central nervous system. Diphtheria toxone also acts specifically upon nervous structures. Diphtheria toxin, on the other hand, is a general protoplasmic poison. These facts are of immense importance in the prevention and cure of certain infections, for a correct understanding of the chemical relation between the poison and the particular cell is of the greatest fundamental and practical value. A realization of this fact has stimulated studies which are now in progress upon the relation between the chemical constitution and the physiological action of various substances—studies which have already borne fruitful and useful results.

Tetanus toxine may combine with certain cells without apparently injuring them. Diphtheria toxine also combines with indifferent structures, such as the connective tissue. There is evidently a wide difference between the power to combine and the power to injure. The power to injure, however, is not always evident, as it depends upon the importance and extent of the cells affected. Thus, tetanus toxine may combine with the leukocytes in such a way as to prevent phagocytosis. This may be demonstrated by injecting tetanus spores washed free of toxine, in which case the spores are taken up by the leukocytes and their development is prevented. If, however, a slight amount of toxine is injected with the spores, the poison inhibits phagocytosis and permits the growth and multiplication of the tetanus microorganisms and the further production of toxin.

From our standpoint the most important property of a true toxine is its power to produce specific antitoxins. This will be given separate consideration.

Ehrlich conceives the toxin to be a complex molecule containing both a haptophore and a toxophore group. The haptophore or seizing group is that part of the molecular structure which combines in a chemical sense with the antitoxin or with the receptors of the cell. The toxophore group is the poisonous part of the toxin molecule. This is usually represented diagrammatically (see Fig. 49, p. 357).

It may readily be demonstrated by simple experiments that the toxophore group is much more unstable than the haptophore group. The toxin may degenerate so that it has little or no poisonous properties left; however, its combining properties remain unaltered. Such a degenerated toxin is known as a *toxoid*. A toxoid, then, is an altered toxin which possesses the combining property of the original toxin, but has lost its poisonous power. Some years ago I proposed to draw a distinction between the terms "toxine" and "toxin." The toxine is the crude filtered culture and contains several poisonous substances as well

as other bodies. The toxin is the specific poison in the toxine. Thus, a filtered broth culture of diphtheria is known as diphtheria toxine. This filtrate contains at least two primary metabolic poisons: *toxin* and *toxone*. The toxin produces the acute symptoms and death; the toxone produces the late paralysis. A filtered broth culture of tetanus is called the tetanus toxine. The filtrate contains at least two primary metabolic poisons: tetanoplasmin and tetanolysin. For a further discussion of the diphtheria and tetanus poisons see page 373.

ANTITOXINS

An antitoxin is an antibody formed in an animal through the stimulation of a specific toxin. The usual method of producing an antitoxin is by the repeated injections of increasing amounts of toxine into a susceptible animal. The strongest antitoxins are obtained from animals that are very susceptible to the toxine, but all susceptible animals by no means produce antitoxins, although repeatedly injected with the appropriate poison. Thus, a guinea pig which is very susceptible to diphtheria will not form diphtheria antitoxin, even after the repeated administration of diphtheria toxine. Guinea pigs are also exceedingly susceptible to tetanus and react characteristically and violently to tetanus toxine, but the repeated injections of subminimal lethal doses of tetanus toxine into a guinea pig do not immunize that animal, nor do they induce the formation of antitoxin. In fact, Knorr and also Behring and Kitashima have shown that guinea pigs develop an increasing sensitiveness to repeated injections of tetanus toxine instead of an increasing resistance. In other words, the guinea pig, a susceptible animal, lacks the mechanism of antitoxin formation which is possessed in such a high degree by horses and other animals. Antitoxin produced by the horse or other animal when injected into the guinea pig will protect it.

On the other hand, insusceptible animals, as a rule, do not produce antitoxin, but there are notable exceptions to this rule. Metchnikoff has shown that the cayman, an animal insusceptible to tetanus, will, however, produce tetanus antitoxin if the animal is kept at an elevated temperature (32° to 37° C.), but not if kept cold (20° C.). The mechanism of antitoxin formation is not understood, and the only way of determining whether a certain species of animal is suitable or not is by experimental trial. There is a very great difference in the ability to produce antitoxin even among different individuals of a suitable species. Thus, some horses have this power developed to such an exquisite degree that they produce a high grade of antitoxin for prolonged periods—years. Other horses cannot be stimulated to antitoxin

production. This difference among horses is well known to manufacturers, who have no means of knowing beforehand which horses will be profitable. The only practical method at present known is to discard those animals which refuse to respond to the stimulation of the toxine injections.

There are several reasons for selecting the horse for the production of immune sera for human use. On account of its size it furnishes large quantities of blood; the serum of the horse is the blandest blood serum of any known species; finally, the horse furnishes antitoxin in higher potency than any other known animal.

Just how and by what cells antitoxins are formed in the body is not known. They are not formed directly from the toxines. In some way the toxine excites the cell to the formation of the antibody. The antibody leaves the cell and becomes "dissolved" in the blood and tissue juices. Perhaps the white blood cells (Metchnikoff), perhaps the connective tissue cells (Ehrlich), are chiefly concerned. Within the body most of the antitoxin is found in the blood, but it also exists in greater or less concentration in practically all the fluids of the body and may also appear in the excretions, as the urine, saliva, milk, and bile.

Nothing definite can be stated concerning the chemical nature of antitoxins. Evidence strongly points to the fact that they belong to the higher proteins. In all probability antitoxins are globulins, at least they come down with the globulins from which they have not been separated.

Antitoxins are somewhat more stable than the toxines; therefore, the standards by which diphtheria and tetanus antitoxins are measured consist of dried and precipitated antitoxins (and not toxines) preserved under special conditions. Further, the toxines have a more complex constitution than the antitoxins. When the toxines deteriorate they change qualitatively as well as quantitatively. The antitoxins have a simpler constitution and deteriorate simply by a loss of power.

Antitoxins are destroyed by heat, acids, and many chemicals. They gradually deteriorate spontaneously when in solution. Thus, Anderson has found that the average yearly loss of the potency of diphtheria antitoxin at room temperature is about 20 per cent.; at 15° C. it loses about 10 per cent.; and at 5° C. about 6 per cent. There is little difference between the keeping qualities of untreated sera and sera concentrated and refined by the Gibson process. Dried diphtheria antitoxin kept in the dark at 5° C. retains its potency practically unimpaired for at least 5½ years. Antitoxic sera should always be kept in a cool, dark place. While antitoxin loses some of its potency with time, and while recently tested sera of known unit value are always desirable, there is absolutely no reason why a serum, however old, should not be employed provided a fresh supply is not at hand. It should be remem-

bered that antitoxins deteriorate quantitatively only, in other words, an old antitoxin is quite as useful in proportion to its unit strength as a fresh serum; in fact, antitoxic sera are frequently two years old when placed upon the market by manufacturers, for the reason that it is believed an old serum is less apt to produce rashes (the serum disease).

Antitoxins are strictly specific; that is, they neutralize the corresponding toxine and have no other apparent action within the body. The occasional ill effects, such as the serum sickness, following the injection of antitoxic sera are due to other substances (the proteins in the serum) and not to the antitoxins themselves.

Antitoxins may be injected subcutaneously, intravenously, into the subarachnoid space, into a nerve, into the brain substance, or into any of the body cavities. Antitoxins are practically useless when given by the mouth, as very little is absorbed. Antitoxins when injected into an organism disappear rather quickly. Some of the antitoxin is bound to the corresponding toxine, if any is present, some combines with the cells, but the greater part is eliminated as antitoxin in the urine, bile, saliva, etc. The antitoxin contained within the organism that produces it actively, as the result of an attack of the disease or as a result of the injection of toxin, disappears much more slowly from the body than when the antitoxin is injected into the organism, as in passive immunity. Passive or antitoxic immunity is, therefore, transient; it cannot be depended upon for more than ten days or two weeks.

When antitoxic serum is injected subcutaneously the antitoxin is absorbed slowly. It requires about 48 hours under these circumstances for the antitoxin to appear in the blood in maximum amount. Therefore, when very prompt action is desired, the antitoxic serum may be introduced directly into the circulation by intravenous injection.

There are a number of antibodies that are either true antitoxins or closely resemble these antibodies. Some of these antibodies neutralize the true bacterial toxines, others the poisons of animal origin, others the poisons of plant origin, and others neutralize the activity of ferments. The principal antitoxins, according to this classification, are brought together as follows:

(1) *Bacteria Antitoxins.*—The three principal and most potent bacterial antitoxins are those of diphtheria, tetanus, and botulismus. The following are also considered to have antitoxic properties: pyocyaneus, symptomatic anthrax, antileukocidin and antilysin against bacterial hemolysins.

(2) *Animal Antitoxins.*—These antitoxins are produced by animal poisons belonging to the venoms. True antibodies are obtained against snake venom and similar poisons in spiders, eels, wasps, scorpions, fish, salamanders, and toads.

(3) *Plant Antitoxins.*—These are antirisin, antiabrin, antirobin, anticrotin, and pollantin, the pollen antitoxin against hay fever.

(4) *Ferment Antitoxins.*—Antibodies may be obtained against ferments, such as pepsin, urease, rosinase, steapsin, trypsin, fibrin ferment, lactase, cyranase; and antibodies may also be obtained against the ferments found in bacterial cultures.

There are comparatively few antitoxic sera of practical use in human therapy, just as there are relatively few true bacterial toxines. The best known antitoxins are those of diphtheria, tetanus, and botulismus. Numerous other antitoxic sera are found upon the market or have been described, but they are of doubtful or no practical value; any power such so-called antitoxic serum may have is due to antibodies other than antitoxins.

Antitoxins are valuable both as curative and immunizing agents. Their preventive action depends upon the fact that they meet the toxin, unite with and neutralize it, thus rendering it harmless. As already stated, the antitoxins remain in the body a brief time and their immunizing power, while of a high grade, is transitory. They disappear in about ten days or two weeks; the immunity must, therefore, be renewed in special cases by repeated injections of the antitoxin until the danger is passed. This phase of the subject is considered in more detail under the prevention of diphtheria and tetanus. The usual immunizing dose for diphtheria is 1,000 units, for tetanus 1,500 units.

As a curative agent antitoxin must be administered early and in sufficient amount to insure success. It is most important to give the antitoxin early—before the damage is done. Too great emphasis cannot be laid upon this point. After the toxin has united with the cells it cannot be dislodged by the antitoxin. The importance of giving antitoxin early is well illustrated in the case of diphtheria. When moderate amounts (3,000 to 10,000 units) are injected on the first day of the disease the mortality is practically nil. The mortality increases with each hour's delay.

The importance of giving this sovereign remedy early is also illustrated in the experiments of Rosenau and Anderson[1] upon the influence of antitoxin upon post-diphtheritic paralysis. It was found that one unit of antitoxin, given not less than 24 hours after a fatal dose of diphtheria toxine in a guinea pig, greatly modified the post-diphtheritic paralysis and saved the life of the animal, whereas 4,000 units given 48 hours after the infection did not modify the paralysis or save the life of the animal. Four thousand units of antitoxin is an enormous amount for a guinea pig weighing about one-half a pound. Weight for weight, it corresponds to 400,000 units for a 50-pound child. The fact that one unit of antitoxin saves life when administered timely,

[1] *Hyg. Lab. Bull.*, No. 38, 1907.

whereas enormous doses fail totally when delayed, should be sufficient to place physicians on their guard; increased dosage cannot atone for delay. When cases are seen late in the progress of the disease it is good practice to give large doses of antitoxin, for the reason that the poison is being elaborated continuously and some of it is free in the circulating blood. The antitoxin unites with and neutralizes the uncombined poison and thus protects the cells against further damage. This refers to tetanus as well as diphtheria. Tetanus antitoxin is a very valuable immunizing agent, but is of less value after symptoms have appeared, for then most of the damage has been done.

Preparation of Antitoxin.—The antitoxin used in human therapy is practically always contained in the blood serum or blood plasma of the horse. The blood is drawn from the jugular vein into sterile bottles. The bleeding should never be done until a week or more has elapsed since the last injection of toxine, so as to allow time for the disappearance of the poison from the circulation. The horses are given no food for about 24 hours preceding the bleeding, so that the blood may not contain the fresh products of digestion and metabolism. After the blood is drawn it may be allowed to clot spontaneously. In the case of horse blood this takes place more quickly at room temperature than in the ice chest. The clot is allowed to contract for a few days and the serum containing the antitoxin is then drawn off with a pipette or simply decanted. In this way a clear transparent serum is obtained which, if protected against contamination by the usual bacteriological precautions, is sterile and may be preserved indefinitely. It is almost a universal practice, however, to add a preservative; either chloroform (0.3 per cent.), phenol (0.5 per cent.), or tricresol (0.4 per cent.). These preservatives in the amounts named are harmless when injected and have practically no effect upon the antitoxin itself. They gradually precipitate the albuminous matter from the serum, which settles as a white amorphous deposit and which may be disregarded, as it is harmless. Chloroform produces a better-looking serum, but the less volatile preservatives are usually preferred on account of their stability and, hence, greater reliability.

By the method of allowing the blood to coagulate, as above described, only about one-third of its volume is recovered as antitoxic serum. A much greater yield may be obtained by citrating the blood: sodium citrate prevents the clotting of blood. A solution of this salt is placed in the bottle which is to receive the blood directly from the horse, in sufficient amount to be present in 1 per cent. of the whole blood. The corpuscles soon settle to the bottom, leaving the clear supernatant plasma, which is then decanted or drawn off with a pipette. In this way the yield of antitoxic fluid is about 90 per cent. of the volume of

the blood, and is, therefore, preferred to the less economical method of allowing the blood to clot.

The citrated plasma may further be "purified" or concentrated by various methods, that generally used being a modification of Gibson's [1] method, based upon the earlier experiments of Atchinson.

Ordinary antitoxic serum contains serum globulins (antitoxic), serum globulins (non-antitoxic), serum albumins (non-antitoxic), serum nucleoproteids (non-antitoxic), cholesterin, lecithin, traces of bile coloring matter, traces of bile salts and acids, traces of inorganic blood salts, and other non-proteid compounds. Refined serum contains serum globulins (antitoxic), traces of serum globulins (non-antitoxic) dissolved in dilute saline solution.

Gibson's Method of Concentrating Diphtheria Antitoxin.—Gibson [2] prepared a refined and concentrated diphtheria antitoxin by first precipitating the antitoxic serum with a half saturation of ammonium sulphate. This throws down the globulins. The precipitate is collected and dissolved in a saturated solution of sodium chlorid. Only a portion of the globulins, but all of the antitoxin, passes into the solution. Through the precipitation by ammonium sulphate and solution in sodium chlorid the nucleoproteins and the insoluble globulins are eliminated. The soluble globulins with the antitoxin are now precipitated by the addition of acetic acid. The precipitate is collected upon a filter, partially dried, and finally placed in a sac of parchment membrane and dialyzed in running water. The resulting fluid is then an antitoxic solution of soluble globulins which is rendered neutral, and sufficient sodium chlorid is added to make it isotonic. In carrying out the process there is a loss of about 30 per cent. of antitoxin units because of retention on filters, loss in dialysis, etc., but the resulting solution of antitoxic globulins has a greater concentration than the original serum from which it was obtained. Thus, a serum containing only 200 units of antitoxin per c. c. may, after concentration with Gibson's method, contain as much as 500 units of antitoxin per c. c.; and one having an original potency of 300 may contain 700 units per c. c. in the final product.

The advantages of the antitoxic globulins are that a smaller bulk is required to give a corresponding number of units of antitoxin. Less foreign proteins are injected, and there is a resulting decrease in the number and severity of those showing the serum sickness. By the method of concentration and refining, antitoxic sera too weak for practical purposes are thus saved.

Dried Antitoxin.—The antitoxic serum or the antitoxic plasma may be dried by any of the methods in common use, care being taken to

[1] *Jour. of Biolog. Chem.*, Vol. I, 1906.
[2] Ibid., Vol. I, Nos. 2 & 3, Jan., 1906, p. 161.

prevent bacterial contamination and also to prevent overheating. The antitoxic fluid is usually dried in shallow layers on pans in a vacuum apparatus, to the form of golden yellow amorphous flakes. These are ground to a powder. About 100 c. c. of serum or plasma yields approximately 10 grams of dried residue. It is, therefore, necessary to redissolve the dried antitoxin in at least 10 parts of normal saline solution. The advantages of antitoxin in the dried form are that when preserved in a cool, dark place it retains its potency practically indefinitely. The only disadvantage is that it goes into solution with some difficulty, and the making of this solution requires not only time, but is rather inconvenient.

Mode of Action.—The mode of action of antitoxins is now fairly well understood. One thing is certain, and that is that the antitoxin unites directly with the toxin. This may be readily demonstrated by adding a little antitoxin to some toxin in a test tube and then injecting the mixture into a susceptible animal; no symptoms result. Diphtheria antitoxin combines with diphtheria toxin more quickly than tetanus antitoxin combines with its poison. Thus, in the case of diphtheria the union between the poison and its antibody is complete in less than twenty minutes at room temperature, while in the case of tetanus it requires one hour. These facts are of practical importance in the work of standardization, in which case the toxines and antitoxins are mixed in the test tube and the combining action must be complete before the mixtures are injected into the test animals in order to insure accurate results.

Ehrlich believes and strongly defends his assumption that an antitoxin unites with a toxin just as an acid unites with an alkali, that is, the one has a strong chemical affinity for the other, and the union is simple and direct. On the other hand, Arrhenius and Madsen insist that, instead of considering the toxine as a complex mixture of various substances, such as a toxin, toxone, etc., it would be simpler to consider it as a single (at least homogeneous) substance which has a very weak affinity for the antitoxin and that in mixtures containing toxine and antitoxin there are always both free toxin and free antitoxin. Arrhenius draws his analogy from known facts in physical chemistry, particularly from studies upon the relation between solutions of boracic acid and ammonia. These two substances have a comparatively weak affinity for each other, and in mixtures all the boracic acid does not combine with all the ammonia, but there are always present both free ammonia and free boracic acid.

When ammonia and boracic acid are brought together in watery solution some of the ammonia at once unites with some of the boracic acid to form ammonium borate. This reaction starts with a certain velocity, but as the mass of ammonium borate increases the velocity of

the reaction gradually diminishes. After a time a condition is reached when the ammonium borate has a maximum value and does not further increase, no matter how long the reaction is allowed to proceed under the given conditions.

When this condition of equilibrium is reached the mass contains a certain quantity of water, ammonia, boracic acid, and ammonium borate; but these substances are not at rest. The ammonia and boracic acid will always react when in the presence of each other, whether or not ammonium borate is present. But, as the appropriate amount of ammonium borate remains constant, it is understood while this continuous association between the ammonia and the boracic acid is going on there is at some time a reversible action—that is, a dissociation of the ammonium borate to reform ammonia and boracic acid. These two reactions take place simultaneously.

Arrhenius believes that the diphtheria poison changes slowly according to the laws of monomolecular reactions, that the toxin combines feebly with the antitoxin, the equilibrium constant being equal for both. The claim, however, that the toxine is a simple substance having a weak affinity for the antitoxin and that the combination of toxin and antitoxin follows the Guldberg-Waage law, and that the reaction is, therefore, reversible, seems untenable in the light of the evidence brought forward by Ehrlich, Nernst, Michaelis, and others.

ENDOTOXINS

In contradistinction to the soluble or exotoxins, there is a group of poisons known as endotoxins. The existence of endotoxins was taken for granted before they were actually demonstrated. As soon as it was found that only some bacteria produce soluble specific toxines it was at once assumed that the other bacteria must contain similar poisons, but closely bound within the cell and insoluble in ordinary culture fluids. It was further assumed that these endotoxins were in some way set loose in the body and thereby produced the lesions and symptoms of the disease. The endotoxins are conceived to be poisons very closely bound up with the protein contents of the bacterial cell, and are liberated in the body when the bacterial cell dies and disintegrates. However, it by no means follows that these endotoxins are poisons similar in action and composition to the soluble true toxins; in fact, there is evidence to indicate the contrary. It is true that some bacteria, such as the dysentery bacillus, cholera vibrio, and a few other microorganisms that produce little or no soluble toxine, may be ground up so that the bacterial cells are mechanically ruptured, thus liberating the endotoxin. In most cases of so-called endotoxic action the reaction of anaphylaxis appears to be the best explanation.

TETANUS TOXINE

On account of its virulence, its solubility, and the characteristic contractions which it induces, the poison of tetanus has been a convenient and favorite subject of investigation. It was the first of the bacterial toxines to give fruitful results in serum therapy. The toxine is readily soluble in the medium in which tetanus grows, whether fluid or solid; it diffuses throughout gelatin or agar. The culture filtered free of all bacterial cells is called the tetanus toxine. This is really a complex substance containing various poisons and other bodies. Two of these poisons in particular have been studied: *tetanospasmin* and *tetanolysin*. It is the former which produces the convulsions characteristic of the disease and concerns us especially. This poison is a type of a true toxine. It is exceedingly poisonous in very small quantities; is readily rendered inert by heat (60° C.); contains both a toxophore and haptophore group; produces antibodies when introduced into susceptible animals, and produces symptoms only after a definite period of incubation. In all these characteristics tetanus toxine resembles diphtheria toxine.

Tetanus toxine is one of the most poisonous substances known. Amounts as small as .000,006 gram of the standard precipitated toxine prepared by me in the Hygienic Laboratory at Washington invariably kills a guinea pig weighing 350 grams in about four days. As this precipitate consists mostly of albumins, peptone, amino acids, volatile substances, ammonium sulphate, and other salts, it will be seen that but a small proportion of the weight consists of pure poison. Brieger and Cohn found that their strongest tetanus toxine killed mice weighing 15 grams when given subcutaneously in doses of .000,000,05 gram, and they calculate that .000,23 gram would be a fatal dose for a man weighing 70 kilograms.

Tetanus toxine is not equally toxic for all species of animals; on the other hand, there is an extraordinary constancy in its toxicity upon individuals of the same species; that is, the same quantity of toxine per gram weight of a particular animal always produces similar results.

Tetanus toxine is harmless when given by the mouth. It is not absorbed from the intact intestinal tract, but is affected by the digestive juices. I have fed guinea pigs with as much as 24,000 and mice 18,000 times the minimal lethal dose of tetanus toxine without producing any apparent ill effects.

All attempts to isolate the specific toxine as a definite chemical compound have proved unavailing. We are totally ignorant of its chemical nature. The only way by which it may be recognized is through its effects upon animals. By this means we are enabled to de-

termine not only the presence of the poison, but also to estimate its concentration in a solution and to watch its deterioration.

Brieger in 1886 isolated from a contaminated growth a basic substance or ptomain which he called "tetanin" ($C_{13}H_3N_2O_4$). Shortly afterward he obtained another ptomain which he named "tetanotoxin" ($C_5H_{11}N$). These substances caused muscular contractions when injected into mice, and Brieger believed them to be the true poison of tetanus. They are now only of historical interest, for the studies of Kitasato and Weyl in 1890 with pure cultures of tetanus found that Brieger's purified extracts did not produce the characteristic symptoms of tetanus in experimental animals.

Brieger and Fraenkel in 1890 obtained an alcoholic precipitate from filtered broth cultures which they termed "toxalbumin" and which had undoubted toxic properties. Hayahsi concludes from his work upon the subject that the toxin isolated according to the Brieger-Boers method, as well as by his own modification, shows a definite albumin reaction. However, this does not prove that the toxin itself is a protein.

The powerful action of the tetanus poison in such minute amounts, its thermolability, and the period of incubation lend countenance to the view that the toxin may be a ferment. There is, therefore, nothing but analogy to class tetanus toxin with the ferments.

Ehrlich, in a parallel work to his researches upon the constitution of diphtheria toxine, showed that tetanus toxine contains both a toxophore and a haptophore group and that the antitoxic immunity is explained by the presence of free receptors in the blood. The receptors combine directly in a chemical sense with the haptophore group, thus neutralizing the poison.

Ehrlich,[1] 1898, definitely proved that tetanus toxin contains at least two poisons: (1) *tetanolysin*, and (2) *tetanospasmin*. He showed that these two poisons do not always appear in the same relative proportion in different preparations. Some of the toxins that have strong tetanic properties have weak hemolytic action, and *vice versa*. The hemolytic affinity of the toxin weakens much quicker than the tetanospasmin. This occurs spontaneously as well as when it is heated to 50° C. for 20 minutes. The two poisons have different combining affinities. If tetanus toxine is brought into contact with red blood corpuscles, the greatest part of the tetanolysin is bound by the red corpuscles, while the tetanospasmin remains in the solution. Each one of these two poisons has its own antitoxin. If several different tetanus sera are examined, it will be found that they have no parallel neutralization for tetanolysin and tetanospasmin. In one particular case Ehrlich found a serum that was strongly antispastic and had practically no antilytic power.

[1] Ehrlich, P.: *Berl. klin. Woch.*, 1898, No. 12.

Bolton and Fisch [1] have shown that the toxine makes its appearance in the blood of the horse several days before any symptoms of tetanus are observed, and that it gradually increases until about two days before symptoms become noticeable, and then it suddenly diminishes and even disappears in some cases. The amount of toxine varies considerably in different horses. In one instance the serum of a horse, about two days before symptoms of tetanus appeared, was sufficient to kill a guinea pig in the dose of 0.1 c. c. The fact that tetanus toxine may appear in such large quantities in the blood without symptoms of tetanus is of very great practical importance in the production of both diphtheria and tetanus antitoxins and other therapeutic sera.

Tetanus toxine is readily destroyed by heat, sunlight, acids, and other agencies. Anderson, in 1907, found that when tetanus toxine is exposed to 5 per cent. formalin for six hours a guinea pig is able to withstand 100 minimal lethal doses of this formalinized poison. Three per cent. formalin after twenty-four hours' exposure destroys the toxine; it destroys a part of the toxine in one hour, its action increasing with the length of exposure. This indicates that formalin should prove a useful antiseptic and antitoxic substance for local application to wounds.

Tetanus toxine is prepared from bouillon cultures grown anaerobically at 37° C. for 6 to 15 days. The culture fluid is then filtered through porcelain or diatomaceous earth; the germ-free filtrate contains the poison. This is used to inject into horses for the purpose of producing tetanus antitoxin. Tetanus toxin in solution is so unstable that it cannot be depended upon for the purpose of accurate tests. From a practical standpoint it is all-important to obtain a stable poison. Herein lies the crux of the problem, so far as the standardization of tetanus toxin is concerned. If soluble poisons are used to measure the value of antitoxic serums, as is the case with the German method, it is found necessary to redetermine through a series of mice the strength of the toxine each time a serum is tested. The fact that tetanus toxine does not exhibit the same constancy as diphtheria toxine in solution has thrown much confusion and no little difficulty into the work of standardizing its antitoxin.

Rosenau and Anderson succeeded in obtaining a dry poison by precipitating it from solution with ammonium sulphate. The excess of salt is removed in the dialyzer. The toxine may be further purified by again bringing it into solution and reprecipitating it several times. This, however, is not necessary in ordinary work. The precipitate is collected and dried in a vacuum over sulphuric acid and preserved in vacuum tubes, under the influence of pentaphosphoric (P_2O_5) acid in a cool, dark place. Under these conditions the poison does not diminish in toxicity during a period of over two years. It loses its toxicity

[1] *Trans. Assoc. of Am. Phys.*, XVII, 1902, pp. 462-467.

rather slowly when exposed to light, heat, and other influences. One of the sealed tubes sent from Washington to Manila arrived there without appreciable loss of strength. It is this dried poison which is distributed to manufacturers and other laboratories engaged in the work of standardizing tetanus antitoxin.

Mode of Action.—It has been known for a very long time that tetanus toxin affects chiefly the central nervous system, but it is only comparatively recently that it has been demonstrated experimentally in what way the poison reaches the nerve centers. For this information we are indebted especially to the work of Marie and Morax,[1] 1902, and Meyer and Ransom,[2] 1903. It is now definitely known that the motor nerves have a specific affinity for tetanus toxin. When the toxin is placed subcutaneously the adjacent motor nerve endings at once begin to take it up and it is then transported in the axis cylinder to the cord. This action may be compared to the absorption of nourishing liquids by the roots of a plant. The lymphatics also absorb much of the toxin and in a short while it appears in the blood stream, which carries it to all parts of the body, where again it is absorbed by the motor nerve endings which are bathed in the toxin-laden fluid. The toxin does not reach the nerve cells directly through the blood, for even after introducing the poison into the subarachnoid space there is a general poisoning and not a cerebral tetanus.

The injection of tetanus toxin into the posterior root of the spinal nerves leads to a *tetanus dolorosus* which is characterized by strictly localized sensitiveness to pain. According to Meyer and Ransom, the reason why the sensory nerves do not play any rôle in the conduction of the poison lies in the presence of the spinal ganglion, which places a bar to the advance of the poison.

Milchner in 1898 showed that tetanus toxin combines chemically with the central nervous system. A direct combination takes place when tetanus toxin and brain substance are mixed in the test tube. This action has been studied by Metchnikoff, Wassermann, and Takaki, Roux and Borrel, Denys, and others. It seems that the phenomenon of the fixation of the tetanus toxin by nervous tissue, in spite of some analogies, cannot be likened to the action of antitoxin on toxin. The toxin at first fixed by the nervous substance again becomes free *in vitro* and *in vivo*. The union between the tetanus toxin and the nervous tissue appears to be a feeble chemical absorption which may be readily dissociated and which does little harm to the toxin. On the other hand, the union between toxin and antitoxin is much more stable and definite.

[1] Marie and Morax: *Annales de l'Institut Pasteur,* XVIII, 1902, p. 818.
[2] Meyer and Ransom: *Archives für experimentelle Pathologie und Pharmacologie,* Vol. 49, 1903.

TETANUS ANTITOXIN

Tetanus antitoxin is contained in the blood serum of horses highly immunized by repeated injections of tetanus toxine. It is necessary to begin with exceedingly small doses, for the reason that horses are very susceptible to tetanus. Time may be gained and accidents avoided by guarding the first few injections with tetanus antitoxin. The injections are given at intervals of about a week and may be rapidly increased so that in a few months a horse will be able to stand several hundred cubic centimeters of an exceedingly strong poison. The horse should never be bled for the purpose of procuring the antitoxic serum until at least two weeks have elapsed since the last injection of the toxine, in order to be sure that all the poison has disappeared from the circulating blood.

Tetanus antitoxin is an antibody which corresponds in all essential respects to diphtheria antitoxin. It neutralizes the poison probably by direct chemical combination whether in the body and in the test tube. In human therapy it is used either by subcutaneous or intravenous injection, by injecting it directly into the large nerve trunk leading from the wound, by placing it in the subarachnoid space, or by injecting it directly into the brain substance. It is also used in dried form as a dusting powder upon the wound. When tetanus antitoxin is administered subcutaneously it is absorbed rather slowly. Knorr found the maximum quantity in the blood only after 24-40 hours, and from this time on the amount steadily decreases, so that by the sixth day only one-third of the actual quantity is present; by the twelfth day only one-fiftieth, and at the end of three weeks no antitoxin whatever could be demonstrated. Hence, it is important to give the first dose in a case of tetanus intravenously.

The specific action of tetanus antitoxin makes it a valuable prophylactic; it has less use as a curative agent, for the reason that after symptoms have appeared most of the damage to the nerve cells has been done. While antitoxin has a limited value as a remedial measure, it is by no means to be neglected, for the reason that it neutralizes the free poison in the circulating blood and elsewhere in the body and thus prevents further damage of the nerve cells. In the use of tetanus antitoxin as a preventive it should be remembered that it is quickly eliminated from the body, so that in wounds which continue to suppurate with foul-smelling pus, especially when the pus contains end-spore-bearing rods, and in all wounds in which there is a suspicion of special danger about 1,500 units of the tetanus antitoxin should be administered at intervals of about ten days.

STANDARDIZATION OF ANTITOXIC SERA

The method of measuring the strength of diphtheria and tetanus antitoxins is exceedingly accurate and satisfactory. The tests are physiological, that is, depend upon animal experimentation. Guinea pigs are used because they are particularly susceptible to both tetanus and diphtheria toxines and react to these poisons so uniformly that they serve the purpose of an accurate analytical balance. In order to obtain precise results it is essential that all the conditions of the test be uniform. It is, therefore, advisable to follow the official methods, which have been prescribed in great detail. All antitoxic sera upon the American market are standardized in accordance with the official unit dispensed by the federal government. This work is done in the Hygienic Laboratory of the Public Health Service.

The Standardization of Diphtheria Antitoxin.—The immunity unit for measuring the strength of diphtheria antitoxin may be defined as the neutralizing power possessed by an arbitrary quantity of diphtheria antitoxic serum kept under special conditions to prevent deterioration in an authorized laboratory.

From a theoretical viewpoint the unit may be defined as that quantity of diphtheria antitoxic serum which will just neutralize 200 minimal lethal doses of a pure poison. By a "pure" poison is understood one containing only *toxin* and no *toxoid, toxone,* or other substance capable of uniting with the antibodies.

The first definition may be compared to the platinoiridium bars kept under special conditions in Paris or Washington as the standard yard or meter. If all the meter bars or yardsticks were lost it would be difficult, if not impossible, to reproduce others having the exact lengths of the originals. These standard measures are, therefore, guarded against deterioration just as the standard antitoxic sera are preserved under strict conditions of light, heat, moisture, etc., in the Hygienic Laboratory of the Public Health Service at Washington. From time to time duplicates of this serum are made to guard against deterioration or accident to the original.

The second definition may be compared to the original conception of the meter, which was intended to be one ten-millionth of the quadrant of a great circle of the earth. Theoretically, therefore, if all the meter bars were lost this unit of measurement could be reproduced with approximate fidelity. In the same way it is theoretically possible to reproduce the diphtheria antitoxic unit in consideration of the fact that it has just 200 combining units.

The test by which the strength of antitoxin is measured is a physiological one, and depends upon the neutralization of the toxin by the

antitoxin. This neutralization can only be determined by injecting the toxine-antitoxin mixtures into guinea pigs and noting the results. The unit for measuring the strength of diphtheria antitoxin is a measure of physiologic strength, not of quantity.

In all the early work on this subject the toxine was used as a basis for measuring the strength of the antitoxin, but as the toxine is a much more complex substance than the antitoxin, and as it is less stable, accurate results were not possible. Ehrlich showed that the antitoxin under certain conditions was permanent both in power of chemically combining with and physiologically neutralizing the toxine. One antitoxin, however, cannot be compared with another antitoxin directly. This can only be done through the toxine.

From a practical standpoint, the following illustration of a test will give a clear conception as to how the unit of strength of a serum is determined.

Example of a Test.—It is first necessary to obtain our official yardstick. This may be done by applying to the Hygienic Laboratory in Washington, where the standard serum is kept in a dry powdered form in vacuum tubes under the influence of pentaphosphoric acid in a cold place and carefully preserved from the light. This powder is dissolved, carefully tested, and sent to the applicant in a glycerinated solution. Each cubic centimeter of a certain dilution of this standard serum contains just 1 unit. Before, however, we can measure the potency of an antitoxic serum of unknown strength it is first necessary to standardize a toxine. This is done by mixing one unit of the standard antitoxic serum with varying quantities of the toxine, as follows:

Mixtures of Antitoxic Serum and Toxine Injected Subcutaneously into Guinea Pigs.	Result.
1 immunity unit + 0.14 c. c. toxine	= No reaction.
" " + 0.15 " "	= No reaction.
" " + 0.16 " "	= Slight congestion at site of injection. [This is the L_0 dose.]
" " + 0.17 " "	= Apparent reaction at site of injection.
" " + 0.18 " "	= Injection and edema at site.
" " + 0.19 " "	= Injection and edema at site; late paralysis.
" " + 0.20 " "	= Sometimes death in 5 or 6 days, sometimes late paralysis.
" " + 0.21 " "	= Always causes acute death about the fourth day. [This is the L_+ dose.]
" " + 0.22 " "	= Acute death usually on second or third day.
" " + 0.23 " "	= Acute death on second day.

From this series we learn that one unit contains just sufficient antitoxin to neutralize 0.16 c. c. of the toxine. This is known as the L_0

dose.[1] By the L_0 dose, then, is meant that quantity of poison which just neutralizes or saturates one immunity unit as shown at the necropsy done 48 hours after the subcutaneous injection of the mixture into the guinea pig. The reaction at the site of inoculation at this examination must be hardly noticeable.

In the above illustration the L_+ dose of this toxine is just 0.21 c. c. By the L_+ dose is meant the smallest quantity of poison that will neutralize one immunity unit plus a quantity necessary to kill the animal on the fourth day. The L_+ dose is the test dose which is used to determine the strength of our unknown antitoxic serum, as follows:

The L_+ (or Test Dose of Toxin) + Varying Amounts of Antitoxin Injected into Guinea Pigs.								Results.
0.21	c. c.	toxine	+ 1/150	c. c.	antitoxic	serum	=	No effect.
"	"	"	+ 1/175	"	"	"	=	No effect.
"	"	"	+ 1/200	"	"	"	=	Late paralysis.
"	"	"	+ 1/225	"	"	"	=	Late paralysis.
"	"	"	+ 1/250	"	"	"	=	Dies 4th day.
"	"	"	+ 1/275	"	"	"	=	Dies 3d day.
"	"	"	+ 1/300	"	"	"	=	Dies 2d day.

From this series it is evident that 1/250 c. c. of the serum contains that amount of antitoxin which will neutralize the toxine in the test dose, leaving sufficient free poison to kill the animal on the fourth day. The serum, therefore, contains one antitoxic unit in 1/250 c. c. of serum. One c. c. of the serum would, therefore, contain 250 units.[2]

Standardization of Tetanus Antitoxin.—There are four methods of measuring the strength of tetanus antitoxin: (1) the German method described by Behring; (2) the French method described by Roux; (3) the Italian method after Tizzoni, and (4) the American method established by Rosenau and Anderson. European standards are admitted to be unsatisfactory and for the most part not accurate. Further, they are complicated and difficult to carry out. The American method, which has been made the official government standard for this and other countries, commends itself for its simplicity, directness, and precision.

The tetanus antitoxic unit is based upon the neutralizing value of an arbitrary quantity of antitoxic serum preserved under special conditions to prevent deterioration in the Hygienic Laboratory of the Public Health Service. This arbitrary quantity now contains ten times the amount of tetanus antitoxin necessary to neutralize somewhat less

[1] L stands for Limit. L_0 stands for the limit of no reaction, and L + the limit of acute death.

[2] For the details for carrying out these tests the reader is referred to the *Hygienic Laboratory Bulletin No. 21* upon "The Immunity Unit for Standardizing Diphtheria Antitoxin," by M. J. Rosenau, which contains the official description and details of the process and its theoretical considerations.

than 100 minimal lethal doses of a standard toxine for a 350-gram guinea pig. That is, 0.1 of a unit mixed with 100 minimal lethal doses of the standard toxine contains just enough free poison in the mixture to kill the guinea pig in four days after subcutaneous injection.

The official definition of a tetanus antitoxic unit is the following: The immunity unit for measuring the strength of tetanus antitoxin shall be ten times the least quantity of antitetanic serum necessary to save the life of a 350-gram guinea pig for 96 hours against the official dose of a standard toxine furnished by the Hygienic Laboratory of the Public Health and Marine Hospital Service.

The standardization of tetanus antitoxin does not differ radically from the standardization of diphtheria antitoxin. The toxins and antitoxins are mixed and the mixture injected into guinea pigs. While, however, the unit is based upon the neutralizing value of an arbitrary quantity of antitoxic serum, the antitoxin is not issued for a basis of comparison, as in the case of diphtheria. A stable precipitated toxine, the test dose of which has been carefully determined, is issued to other laboratories for the purpose of testing.

The value of an unknown serum is measured directly from this standard precipitated toxine, the L_+, or test dose, of which is stated. The L_+, or test dose, of the particular toxin now dispensed by the government contains just 100 minimal lethal doses for a 350-gram guinea pig. This particular toxine is very stable and has not changed appreciably in two years. As soon as it alters or is exhausted the next toxine that will be issued may contain more or less than 100 minimal lethal doses, but the test dose will contain precisely the same neutralizing power.

The tetanus antitoxic unit may be better understood from an example of a test.

An Example of a Test.—Carefully tare a weighing bottle, then add approximately 20 to 50 mg. of the dried poison. Again carefully weigh. Dissolve the toxine in the weighing bottle with salt solution (0.85) in the proportion of 0.1 gram of the dried poison to 166.66 c. c. of the salt solution. This proportion is used for the reason that each cubic centimeter of this solution will represent 0.000,6 gm. of the original dried poison (=100 MLD's). This proportion is taken because it is very convenient in measuring out the test dose, which represents 1 c. c. of the solution. Thus:

44.5692 gm., bottle + toxine.
44.5300 gm., bottle.

.0392 gm., toxine.

0.1 gm. : 166.66 c. c. :: 0.0392 : x.

x = 65.33 c. c.

In other words, if the quantity of toxine placed in the weighing bottle should weigh, as in this instance, just 0.0392 gm., carefully deliver from an accurately graduated burette just 65.33 c. c. salt solution into the weighing bottle; and, as before stated, each cubic centimeter of this solution will be the L_+ or test dose.

Now dissolve the serum of unknown value in accordance with the table of dilutions, and mix aliquot parts of the serum with the test dose of toxine, as follows:

No. of guinea pig	Weight of guinea pig (grams)	Subcutaneous injection of a mixture of—		Time of death
		Toxine (test dose)	Antitoxin	
		Gram.	*c. c.*	
1.....	350	0.0006	0.001	2 days, 4 hours.
2.....	350	.0006	.0015	4 days, 1 hour.
3.....	350	.0006	.002	Symptoms.
4.....	350	.0006	.0025	Slight symptoms.
5.....	350	.0006	.003	No symptoms.

According to this series the guinea pig which received the mixture containing 0.0015 c. c. of the serum died in four days and one hour. Therefore, 0.0015 c. c. of the serum contains one-tenth of an immunity unit, as the unit has been defined as ten times the least amount of antitetanic serum necessary to save the life of a 350-gram guinea pig 96 hours against the official test dose. This serum would, therefore, contain just 66 units per c. c.

METHODS.—In order to obtain reliable and comparable results, it is necessary to take into account all the factors concerned—the composition of the poisons, their concentration, the diluting fluid, length of time the mixtures are allowed to stand, the site of inoculation, etc.

ESTABLISHMENTS LICENSED FOR THE PROPAGATION AND SALE OF VIRUSES, SERUMS, TOXINS, AND ANALOGOUS PRODUCTS.

The following table contains a list of the establishments holding on July 1, 1911, licenses issued by the Treasury Department in accordance with the act of Congress approved July 1, 1902, entitled "An act to regulate the sale of viruses, serums, toxins, and analogous products in the District of Columbia, to regulate interstate traffic in said articles, and for other purposes."

The number of the license of each firm is also given, together with the names of the several products for which licenses have been granted.

No. of license	Establishments	Products
1	Parke Davis & Co., Detroit, Mich.	Diphtheria antitoxin, antigonococcic serum, antistreptococcic serum, antitetanic serum, antitubercle serum, bacterial vaccines, erysipelas and prodigiosus toxines (Coley), tuberculins, and vaccine virus.
2	H. K. Mulford Co., Philadelphia, Pa.	Diphtheria antitoxin, antidysenteric serum, antigonococcic serum, antimeningococcic serum, antipneumonic serum, antistreptococcic serum, antitetanic serum, tuberculins, vaccine virus, bacterial vaccines, normal horse serum, and rabies virus.
3	Dr. H. M. Alexander & Co., Marietta, Pa.	Diphtheria antitoxin, antirabic virus, tuberculins, vaccine virus, and normal horse serum.
5	Fluid Vaccine Co., Milwaukee, Wis.	Vaccine virus.
8	The Cutter Laboratory, Berkeley, Cal.	Diphtheria antitoxin, antistreptococcic serum, tuberculins, bacterial vaccines, and vaccine virus.
9	Frederick Stearns & Co., Detroit, Mich.	Diphtheria antitoxin, streptolytic serum, and pneumolytic serum.
11	Pasteur Institute of Paris, Paris, France.	Diphtheria antitoxin, antidysenteric serum, antimeningococcic serum, antiplague serum, antistreptococcic serum, sérum antivenimeux, antitetanic serum, and antiplague vaccine.
12	Chemische Fabrik auf Actien, Berlin, Germany.	Diphtheria antitoxin and antistreptococcic serum.
14	Health Department of the City of New York.	Diphtheria antitoxin, antitetanic serum, antirabic virus, vaccine virus, tuberculin, and antimeningococcic serum.
15	Dr. W. R. Hubbert Serum Laboratory, Detroit, Mich.	Diphtheria antitoxin.
16	National Vaccine and Antitoxin Institute, Washington, D. C.	Diphtheria antitoxin, antigonococcic vaccine, vaccine virus, normal horse serum, antistaphylococcic vaccine and antistreptococcic vaccine.
17	Lederle Antitoxin Laboratories, New York City.	Diphtheria antitoxin, antistreptococcic serum, antitetanic serum, suspension of lactic acid bacilli, vaccine virus, and antityphoid vaccine.
18	Burroughs, Wellcome & Co., London, England.	Diphtheria antitoxin, antigonococcic serum, antidysenteric serum, anticolon bacillus serum, antistaphylococcic serum, antistreptococcic serum, antityphoid serum, tuberculins, and bacterial vaccines.
19	Memorial Institute for Infectious Diseases, Chicago, Ill.	Diphtheria antitoxin.
21	Swiss Serum and Vaccine Institute, Berne, Switzerland.	Diphtheria antitoxin, antidysenteric serum, antimeningococcic serum, antipneumonic serum, antiplague serum, antistreptococcic serum, tuberculins, anticholera vaccine, antiplague vaccine, antityphoid vaccine, and antitetanic serum.
22	Institut Bacteriologique Lyon, Lyons, France.	Antidiphtheric serum and normal goat serum.
23	Bacterio-Therapeutic Laboratory, Asheville, N. C.	Tuberculins.

No. of license	Establishments.	Products
24	Farbwerke, vormals Meister Lucius und Brüning, Hoechst-on-Main, Germany.	Diphtheria antitoxin, antidysenteric serum, antimeningococcic serum, antipneumonic serum, antistreptococcic serum, antitetanic serum, and tuberculins.
25	Tuberculin Society of St. Petersburg, St. Petersburg, Russia.	Tuberculinum purum.
27	Institut Pasteur de Lille, Lille, France.	Serum antivenimeux.
28	Bacteriologisches Institut Lingner, Dresden, Germany.	Pyocyanase.
29	The Behringwerk, Marburg, Germany.	Antitetanic serum and tuberculin.
30	Dr. G. H. Sherman, Detroit, Mich.	Bacterial vaccines.
31	E. Merck, Darmstadt, Germany.	Antidiphtheric serum, antimeningococcic serum, antipneumonic serum, antistreptococcic serum, normal horse serum (dried), and normal horse serum.
32	Kalle & Co., Biebrich, Germany.	Tuberculin (Rosenbach).
33	American Biologic Co., Kansas City, Mo.	Antirabic virus.
34	The Beraneck Laboratory, Neuchatel, Switzerland.	Tuberculin (Beraneck).
35	Dr. Carl Spengler, Davos-Platz, Switzerland.	I. K. immune blood.

PHAGOCYTOSIS

Metchnikoff gave us the first physical explanation of immunity through his brilliant studies upon phagocytosis. Metchnikoff is a biologist, and as a result of his stimulating observations upon the phagocytes in all the orders of the animal kingdom he has contributed much to our knowledge, not alone of immunity, but to our fundamental knowledge of nutrition and inflammation. The ingenuity and fertility of his views caused a flood of work from others upon these basic subjects in medical biology.

Phagocytosis is a process common to all cells having amebic motion. A phagocyte is any cell capable of absorbing particulate matter into its substance. The process is best seen with an ameba under the microscope.

For a clear understanding of phagocytosis it is necessary to consider three phases of the process: (1) the approach, (2) the engulfment, and (3) the digestion.

The *approach* or *chemotaxis* is a phenomenon which is displayed by almost all motile and unicellular organisms, whether animal or vegetable, as well as by the leukocytes. It manifests itself by a movement of the unicellular organism or the phagocytic cell toward the

particle and seems to be a response to a chemical stimulus. Chemotaxis is said to be *positive* when the leukocytes are quickly and energetically attracted to a substance, and *negative* when this attraction is lacking. There is considerable doubt whether there is true negative chemotaxis in the sense of repulsion. The degree of chemotaxis possessed by any substance may readily be determined by placing it in a capillary tube closed at one end and then inserting the open end of the tube into the tissue of an animal or into a fluid containing active phagocytes. If the substance has positive chemotactic power the phagocytes soon approach the free end of the capillary tube, which they enter; if the substance has negative chemotactic power the phagocytes are not attracted and do not enter the capillary tube. As Emery points out, the leukocytes are in many cases attracted into an infected area to their own undoing, and it must not be forgotten that "even in inflammatory processes which are mild in nature and favorable in result the number of leukocytes which may be killed in the conflict is enormous. The leukocytes are not independent protozoa inhabiting the blood and tissues, but an integral part of the organism. It is to the advantage of the latter that the former should be attracted at once to the seat of invasion, and hence the processes of evolution have led to the development of this function in the nomadic cells of the body. These are extraordinarily susceptible to chemotactic influences. They seem to be attracted by any deviation from the normal situation of the tissues and fluids—a slight injury, a hemorrhage, the presence of a poison, or a foreign body of any sort, or any dead or useless tissue —and the leukocytes are immediately attracted into the area affected. The more we regard the process the more we must regard it as one of the most exquisite examples of means to ends met with in the animal economy."

The engulfment of the bacteria may readily be studied in amebæ in their free living stage. The protoplasm of the ameba is thrown out in the form of pseudopodia; these encircle the particle, which soon appears within the substance of the ameba. The engulfment of particles by the leukocytes and other cells is precisely the same.

The digestion within the cell is entirely comparable to gastric digestion in higher animals. It is now known that active proteolytic ferments dissolve the albuminous particles, and that this takes place in an acid medium may be demonstrated by the use of delicate indicators, such as neutral red.

The phagocytes may take up and digest either live or dead bacteria; they are not simply scavengers. They engulf particles of all kinds, both organic and inorganic. Thus, in anthracosis the particles of coal are mainly carried and contained in the phagocytic cells. The phagocytes play a similar rôle with the malarial pigment, with the granules

of pigment left after a hemorrhage, and with other foreign particles in the body. Phagocytes are also enabled to absorb colloidal substances and fluids as well as particulate matter. They are enabled to dispose of comparatively large masses by removing it piecemeal. Thus, the "core" of boils is gradually removed mainly by the phagocytes. Catgut and silk ligatures are similarly removed and the absorption of the tadpole's tail is disposed of through the same process.

Metchnikoff divides the phagocytes into free and fixed, macrophages and microphages.

The free phagocytes are the leukocytes, lymphocytes, and other blood cells, as the myelocytes from the bone marrow. The fixed phagocytes are the connective tissue cells and endothelial cells. The free phagocytes, according to Metchnikoff, play the more important rôle.

The microphages or microcytes are the mononuclear leukocytes, the polymorphonuclear leukocytes, and the wandering connective tissue cells. The macrophages or macrocytes are the large lymphocytes, the mononuclear pulp cells of the spleen and bone marrow, endothelial cells of the large vessels, and Kupfer's stellate cells of the liver. The microphages play an active part in all acute infections. They are the first to come in the field and for the most part are vegetarians, that is, they take up bacteria especially. The macrophages, on the other hand, are carnivorous, engulfing other cells and protozoon parasites, and are especially concerned in chronic inflammations, such as tuberculosis and leprosy, rather than in the acute processes. These distinctions between the free and fixed phagocytes, the microphages and macrophages, are entirely arbitrary. All the leukocytes have the power of phagocytosis, though in varying degree. This is readily seen in an opsonic preparation or in an examination of a smear of gonorrheal pus, when some of the polymorphonuclear leukocytes will be loaded with the cocci while others contain few or none. The small phagocytes (microcytes) are able to engulf protozoa and animal cells as well as bacteria.

Metchnikoff has insisted since the beginning of his studies upon phagocytosis that this process plays an important, if not the sole, rôle in immunity. He conceives that a true battle takes place between the cells and the invading germs. When phagocytosis is active and successful, immunity is the result. If phagocytosis is absent, or the phagocytes are unsuccessful, the result is susceptibility instead of immunity. Metchnikoff first studied the protective power of the phagocytes in a fresh water crustacean, the daphnia, which, from its transparency and small size, is a very suitable creature for observation. He found that the daphnia is subject to a disease due to the invasion of its body cavity by the spores of a yeast (Monospora), and that if these spores gain access in large numbers they multiply, form into mature organisms, and finally kill their host. When, however, a few spores gain

access he found the leukocytes of the daphnia approach them, form a wall around them, and finally digest and destroy them. It is obvious, therefore, that the immunity of the daphnia to this infection depends upon the activity of its leukocytes. Analogous instances are found in many other animals, including man. In the streptococcus infections particularly Metchnikoff believes their virulence depended upon the absence of phagocytic action.

It soon became evident to Metchnikoff himself that the mechanism of immunity was a much more complicated process than could be accounted for simply by the number and physical activity of the phagocytes. The simple act of phagocytosis alone could not explain all the phenomena. It, therefore, became necessary to study the processes of digestion and the products of excretion of the phagocytes. It soon became evident that the digestive power of the phagocytes is a very powerful one, and substances usually deemed entirely insoluble may be gradually removed by their action. Metchnikoff considers two of these substances to be concerned in immunity: the microcytase and the macrocytase.

The *microcytase* is a ferment-like substance obtained from the microcytes. It is thermolabile and corresponds in all essential respects to the alexin of Buchner or the complement of Ehrlich.

The *macrocytase* is a thermostable substance obtained from the macrocytes. It is concerned with specific acquired immunity. The macrocyte fastens itself to the bacteria, hence was called by Metchnikoff the fixator. It is similar in all essential respects to the "substance sensibilitrice" of Bordet, or the amboceptor of Ehrlich.

Buchner, as well as most other unprejudiced students in immunology, takes the middle ground between the doctrines of the cellular theory represented by Metchnikoff and his school and the doctrines of the humoral theory represented by Ehrlich. It now seems quite evident that both the cells and the body fluids play an important rôle in the mechanism of immunity. It is also equally evident that the mechanism of immunity differs widely with different infections; in some phagocytosis plays a dominant part; in others it seems that the fluids of the body are chiefly concerned. It must not be forgotten that even where the fluids of the body are the chief actors the antibodies are probably in all cases derived from the cells. Just what cells—whether the fixed tissue cells or the free phagocytes—are chiefly concerned in the production of these antibodies is not quite clear.

All observers are agreed upon one thought, and that is, fundamentally immunity is closely allied to the processes of cell nutrition. The receptors of Ehrlich are the mouths of the cells for food. The phagocytosis of Metchnikoff is primarily a mechanism by which cells possessing amebic motion obtain their food. Anaphylaxis, which offers

another explanation of immunity to certain infections, deals with the fundamental problems of protein metabolism. It is, therefore, plain that any experimental research that gives a deeper insight into protein metabolism as well as the more direct researches in immunology has a fundamental bearing upon the prevention and cure of disease.

OPSONINS

The name opsonin (opsono: I cater for, I prepare) is given to substances which occur in the blood and which have the power of preparing bacteria and other cells for ingestion by the leukocytes. The opsonins combine with the bacteria and in that way prepare them for being taken up more easily by the phagocytic cells. In the absence of opsonins, phagocytosis does not take place, and their great importance is, therefore, at once manifest. There is now no doubt concerning the existence of these substances, and the brilliant work of Wright has stimulated a flood of researches which have thrown much light upon this chapter in immunology.

The opsonins are normally present in the blood or may be increased or diminished in amount by the injection of bacteria or appropriate antigen. The opsonins are specific, that is, the blood serum may contain opsonins which prepare staphylococci for the phagocytes, but may contain no suitable substance to prepare streptococci, tubercle bacilli, or some other microorganism. The opsonins are probably similar to the bacteriotropins; their chemical nature, however, in common with other antibodies, is not understood.

The Opsonic Index.—Sir Almroth Wright has modified Leishmann's method for measuring the opsonic power of the blood serum, but the method is somewhat complicated and gives variable results even in the hands of trained workers. It may be questioned whether any of the tests now in use are a true index of the amount of opsonins in the serum, although they may be taken to indicate roughly the measure of their activity. The opsonic index has been especially used as a guide to vaccine therapy rather than in preventive medicine. If, however, we had a satisfactory and ready method by which the specific opsonins of the blood could be measured so that deficiencies could be readily determined and strengthened, we would theoretically at least have a valuable addition to prophylaxis.

LYSINS

Lysins are substances that have the power of disintegrating or dissolving cells or other organized structures. Those that dissolve bacteria are known as the bacteriolysins, those that dissolve red blood

cells are called hemolysins, those that dissolve epithelial or other body cells are called cytolysins or cytotoxins. The lysins in themselves are not poisonous, but through their action they liberate or generate toxic substances and thus play an important rôle not only in the pathogenesis of many infectious diseases and diseased states, but also in their cure and prevention.

Normally the blood possesses bactericidal properties, and it is believed that this is almost entirely due to its power of dissolving the bacterial cells. The bacteriolytic property of normal blood serum is not specific, whereas the bacteriolysins induced through special processes by immunization are strictly specific. The fact that the blood has the power of resisting decomposition longer than other animal fluids was known to Hunter before the era of bacteriology. It was also early known that this property of the blood diminishes spontaneously after it was shed and could be destroyed by heat—about 55° C. The bacteriolytic substances in the blood were first studied by Buchner and Nuttall, who called them alexins. When it was discovered that the blood possesses marked powers of destroying bacteria the conclusion was naturally drawn that herein lies the explanation of immunity. It was soon learned, however, that, though the blood of certain animals may possess marked bactericidal properties, nevertheless they are very susceptible; and, further, that the power to kill bacteria is much more marked in the serum than in the circulating blood in the animal. Thus, according to Lubarsch, 16,000 virulent bacilli will kill a rabbit if injected intravenously; that is, the blood within the body has not the power of killing this number, yet 1 c. c. of fresh blood serum will destroy this number or more in a test tube.

Rabbits are very susceptible to anthrax, although the blood serum of these animals possesses marked bactericidal properties for the anthrax bacillus; on the other hand, the dog is very resistant to anthrax, despite the fact that its blood serum is very slightly bactericidal.

The bacteriolysins were discovered by Richard Pfeiffer[1] in his attempt to actively immunize animals against cholera by the injection of live cultures. He observed that the cholera organisms were disintegrated and dissolved in the peritoneal cavity of the immunized animals. This gave rise to what is now known as Pfeiffer's phenomenon, which, on account of its importance, must be considered.

Pfeiffer's Phenomenon.—Guinea pigs are immunized by the subcutaneous injection of increasing doses of a cholera culture about once a week until they are able to withstand large amounts of a fresh virulent strain. This usually required at least three or four injections. Some of the live microorganisms are now injected into the peritoneal cavity of the immunized animal, and from time to time minute drops

[1] *Zeit. f. Hyg.*, Vol. XVIII, and *Deutsche med. Wochen.*, 1896, pp. 97, 119.

of this injected material with the peritoneal exudate are withdrawn by means of capillary tubes and examined under the microscope. It will be found that the bacteria previously actively motile soon lose their power of motion and die. They then become somewhat swollen and agglutinate into balls or clumps, which gradually become paler and paler. The disintegrating bacterial cells become granular and finally are completely dissolved in the peritoneal fluid. This process usually takes about twenty minutes, provided the animal has been sufficiently highly immunized. For a control a like quantity of the cholera culture is injected into the peritoneal cavity of a normal guinea pig. In this case the microorganisms are not immobilized, agglutinated, or dissolved. Further, the immunized animal remains unaffected while the control animal dies as a result of the infection. This reaction is specific, that is, a guinea pig immunized against cholera will immobilize, agglutinate, and dissolve only the cholera vibrios; a guinea pig immunized with typhoid will act upon typhoid and not upon cholera.

It was soon discovered by Bordet that this reaction takes place not only in the peritoneal cavity of the immunized animal, but will occur in the test tube when the peritoneal exudate or the blood serum of the immunized animal is mixed with the cholera organisms. It was through a study of this reaction that Pfeiffer and Kolle and later Gruber and then Widal discovered and described the ability of blood serum to clump or agglutinate bacteria. It seems evident that this power of the blood serum or the peritoneal exudate of the immunized guinea pig is an important factor in the mechanism of its immunity.

Bacteriolysins are absolutely distinct from antitoxins and agglutinins. Even when these three substances coexist they may be distinguished one from the other through physical, chemical, or biological tests. Nothing is known as to their chemical composition.

Any general statement concerning the thermal death point or other characters of the lysins must be misleading, from the fact that we now know that lytic action is always due to a combination of two substances: one stable, the other unstable; one readily destroyed by heat, the other quite resistant to heat. This important observation was made by Bordet, who was the first to show that two substances are necessary for the phenomenon of bacteriolysis. He considered that one of these substances sensitized the bacteria, and, therefore, called it the "substance sensibilitrice"; this substance is thermolabile. The other substance, which is thermostable, he continued to call alexin. Bordet found that all the essential features of bacteriolysis could be reproduced exactly if red blood corpuscles were substituted for the bacteria. It was this analogy between bacteriolysis and hemolysis that led Ehrlich to an investigation of the latter phenomenon, and his researches led to much

new light upon the subject. Ehrlich introduced new names for the substances which Bordet has shown to be necessary for the phenomenon, and applied his side-chain theory to explain the reaction.

Many names have been given to the two substances which take part in lysis. The thermostable substance has been called substance sensibilitrice, or simply sensibilitrice, immune body, amboceptor, fixator, intermediary body, interbody, philocytase, immunisin, desmon, copula, and preparator; while the thermolabile substance has been called the alexin, complement, addiment, and cytase. We shall speak of the first as the immune body and the second as the complement.

One of the remarkable facts connected with the phenomenon of the lytic poisons is that the poison itself (complement) is normally present in the blood. This substance is a fragile body, readily destroyed at a moderate temperature—55° C. It disappears spontaneously from the serum when kept for a few days; it is destroyed by acids and alkalies and is not specific in its action. Complement appears to be formed by the breaking down of the leukocytes, which accounts for the fact that blood serum after clotting is much more potent than the whole blood; further, complement is absent from fluids containing no leukocytes, such as the aqueous humor.

According to Ehrlich, the immune body has two combining affinities, and, therefore, he called it the amboceptor. It unites on the one hand with the complement and on the other with the receptor of the cell. Bordet, however, considers that the cell unites directly but separately with both the complement and the immune body. The immune body is stable and specific; it is more stable than the agglutinins or even the antitoxins. It is not injured by heating to 60° C., it is weakened at 70° C., and finally destroyed by prolonged exposure at this temperature. It is called the immune body because, according to Ehrlich's views, immunity can only be obtained through it on account of its specific reaction.

In bacteriolytic immunity it is the immune body rather than the complement that is increased.

Just what service the lysins are in the mechanism of immunity is not clear. Recent studies indicate that they may at times be harmful as well as useful. Thus, by dissolving the bacterial cell they have the power of releasing "endotoxins."

The studies upon anaphylaxis have thrown much collateral light upon the probable action of the bacteriolysins in the pathogenesis, cure, and prevention of disease. When the bacteria are dissolved within the body the protein matter which they contain is set free. This may not be poisonous in itself, that is, may not have any of the properties ordinarily attributed to the endotoxins. This foreign bacterial protein, however, may sensitize the organism so that the second time the pro-

tein is liberated it may cause a reaction which may account for some of the pathogenic effects and symptoms of the disease. Buxton and Coleman explain the pathogenesis of typhoid fever as largely due to a solution of the typhoid bacilli within the body, and it is probable that in pneumonia and other infections a like action takes place. An organism that has once reacted to a particular bacterium remains immune so long as it possesses an altered power of reaction, when brought in association with the microorganism in question. Immunity in this sense is an example of allergie and is discussed more in detail under anaphylaxis.

HEMOLYSIS

The hemolysins are substances that lake the blood; that is, they dissolve the hemoglobin from the red blood corpuscle and set it free in solution. A certain part of the stroma of the red corpuscle is also destroyed in complete hemolysis. Some of the hemolysins are specific and others are not. Thus, distilled water will dissolve the hemoglobin from the red corpuscles of almost all animals. Other known non-specific hemolytic substances are various alkalies and acids; plant poisons, such as recin and abrin; bacterial poisons, such as tetanolysin and staphylolysin; and animal poisons, such as snake venom, scorpion venom, etc. The specific hemolysins are obtained by treating (i. e., immunizing) one animal species with the blood corpuscles of another. For example, the blood corpuscles of a guinea pig are injected into a rabbit. After several such injections the blood serum of the rabbit will contain hemolytic substances for the guinea pig's corpuscles. The corpuscles used for immunization are obtained by drawing the blood of the animal into isotonic salt solution (0.85 per cent.) containing about 1 per cent. of sodium citrate, which prevents coagulation. The citrated blood is then centrifugalized, the supernatant fluid drawn off and replaced with isotonic salt solution. This process is repeated three or four times and is known as washing the corpuscles. The object is to remove all trace of serum containing complement and other substances. If this is not done the results will be unnecessarily complicated and misleading. The washed corpuscles are injected into the peritoneal cavity about once a week or ten days until the blood contains the desired hemolytic action. When this point is reached can only be determined by withdrawing small quantities of the blood and testing it.

Hemolytic tests are made by adding together the complement and the immune bodies. The corpuscles are obtained as above described, washed three or four times, and suspended in isotonic salt solution, so that they are present in the proportion of about 5 to 10 per cent. by volume of the salt solution. One c. c. of this suspension is placed in a

small test tube. To this is then added the immune body contained in the serum of the animal that had been injected with the corpuscles. This immune serum is first heated to 55° or 56° C. for one hour in order to destroy the complement. This degree of heat does not injure the immune body. Uniform amounts of the complement are obtained by adding a definite quantity (0.2 of a c. c.) of fresh serum to each test tube. Each test tube then contains a uniform quantity of the corpuscles to be tested, a uniform quantity of complement in the fresh serum, and a variable quantity of heated immune serum containing the immune body. In most cases normal saline solution is added to bring the whole up to a definite volume—say 5 c. c.

These mixtures are now incubated at 37° C. for two hours, being stirred or shaken once or twice in the meantime. The test tubes are now removed and placed in a vertical position in the ice chest from 12 to 24 hours and then examined. If no hemolysis has taken place the supernatant fluid will be untinged and the corpuscles will have settled in a distinct layer at the bottom. If there is complete hemolysis the fluid will be deeply and uniformly colored and there will be no sediment or only a minute deposit of stromata. If the reaction is partial, the fluid will be less deeply colored and there will be more or less of a deposit of undissolved corpuscles. It must be remembered that many bacteria produce hemolysis and that, if the mixtures of corpuscles and sera be incubated for long periods, fallacies may arise from such contaminations.

CYTOTOXINS

If instead of red blood cells an animal is treated with the body cells or glandular cells of another species, it develops the power to dissolve the cells in question. This power is contained in the blood serum and is brought about by substances known as cytotoxins, which are entirely similar to the bacteriolysins, the hemolysins, and other lytic substances. Cytotoxins have been obtained with the spleen (leukocidin), with the sperm (spermotoxin), liver cells (hepatotoxin), kidney cells (nephrotoxin), gastric mucosa (gastrotoxin), placental tissue (syncytiolysin or placentolysin), prostatic tissue (prostatolysin), brain (neurotoxin), and other organs and tissues. When the cytotoxins were discovered they aroused great enthusiasm in the hope that it would now be possible to dissolve and destroy such foreign cells as cancer and other tumors, and pathological processes in which it is desirable to get rid of certain cellular elements. The practical results have been exceedingly disappointing, as further investigations have shown that these cytotoxins are exceedingly weak and, further, are not very specific.

THE BORDET-GENGOU PHENOMENON—FIXATION OF COMPLEMENT

Bordet and Gengou[1] found that bacteria and also red blood cells could be "sensitized" by placing them in heated immune serum. The immune serum is heated to 55° or 56° C. in order to destroy the complement, leaving only the thermostable "substance sensibilitrice" which unites with the bacteria or the red blood cells, and thus prepares or sensitizes them to the action of the complement. If, now, these sensitized bacteria or red corpuscles are added to fresh serum, all the complement contained in the fresh serum is removed or fixed so that the fluid will no longer dissolve bacteria or cells. These facts are of very great importance, and upon them are based the Wassermann reaction for syphilis and other practical applications in immunology.

The *Wassermann reaction* for syphilis is a special method of application of the Bordet-Gengou phenomenon.

The antigen is a watery extract of the syphilitic liver of a case of hereditary syphilis which contains the *Treponema pallidum* in great numbers; usually about 0.1 to 0.2 c. c. of the liver extract is used. Lecithin and other fatty-like bodies may also act as antigen.

The complement is obtained from some fresh guinea pig serum; about 0.2 c. c. is used.

The antibody is usually the unknown quantity, that is, the blood serum of the patient to be tested. This serum is heated to 55° C. to destroy the complement and then diluted in the proportion of 1 to 20 or 1 to 40 with normal saline solution, and 1 c. c. of the dilution is used. The amount must be determined by preliminary tests with known syphilitic serum.

The antigen, the unknown serum, and the complement are then mixed in proper proportions in a test tube and incubated at 37° C. for one hour, at the end of which time all the complement will be removed from the fluid if syphilitic antigen is present in the unknown serum that has been used.

The hemolytic system is then added to the mixture. The hemolytic system usually consists of sheep's corpuscles sensitized with the heated blood serum of a rabbit which has been injected with sheep's corpuscles. These corpuscles then only need the addition of complement to produce hemolysis. The corpuscles are washed and suspended in normal salt solution, to which the heated immune serum (serum of rabbit which has been treated with sheep's corpuscles) is added.

[1] Bordet: *Ann. de l'Inst. Pasteur*, Vol. XIV, 1900, p. 257; Vol. XV, 1901, p. 289.
Gengou: *Ann. de l'Inst. Pasteur*, Vol. XVI, 1902, p. 734.
Bordet and Gengou: *Compte rendu. Acad.*, Vol. CXXXVII, p. 351.

The whole mixture containing the antigen, the antibody, the complement, and the hemolytic system is incubated at 37° C. for two hours, occasionally shaking and stirring the mixture, and is then placed in the ice chest and the result read after 12 to 24 hours. A positive reaction, showing the presence of syphilitic antibody, is determined by the absence of hemolysis. The presence of hemolysis indicates the absence of the specific antibody. Control tests are always necessary, especially to determine that the corpuscles will be completely dissolved by the heated immune serum (antibody) and the guinea pig serum (complement) if the other two ingredients are not added, and there should be no hemolysis if all the substances except the fresh guinea pig serum are used.

The reaction of fixation based upon the work of Bordet and Gengou has many useful practical applications in addition to the Wassermann reaction for the diagnosis of syphilis. If either the antigen or the antibody are unknown, their presence may be determined through the reaction of fixation, because it is strictly specific. The problem is something like the theorem in geometry with the triangle; two sides and an angle of a triangle being known, the other side and angles may be determined.

The antigen is any substance which, when injected into a suitable animal, has the power of generating as antibody. Practically all pathogenic bacteria and pathogenic protozoa act as antigens; many albuminous bodies, such as the venoms, the enzymes, and bland proteins, may also act as antigens. As the reaction is specific, it is possible to determine whether a particular microorganism is the true cause of a disease or not. Thus, Bordet was enabled to satisfy himself that the bacillus which he isolated during the early stages of whooping-cough was the true cause of that disease, as it gave the reaction of fixation with a specific antibody. On the other hand, if the antigen is known, the diagnosis may be made through the reaction of fixation, as in the case of syphilis and the Wassermann reaction.

THE NEISSER–WECHSBERG PHENOMENON OR DEVIATION OF THE COMPLEMENT

Neisser and Wechsberg in 1901 [1] found that, although the addition of a small amount of immune serum renders normal serum more bactericidal or increases its power of protection, a greater addition robs it of most, and sometimes of all, of its bactericidal power. In other words, the solvent effect of the immune body on cells or bacteria in the presence of complement diminishes as an excess of the immune

[1] *Münch. med. Wochenschr.*, 1901, No. 18.

body is added. This particular action is explained by Neisser and Wechsberg as due to a locking up (*ablenkung*) or deviation of the complement which is brought about by an excess of the immune body. The phenomenon is better understood from a study of an example given by Neisser:

(1) Bacteria+10 units immune serum (i. e., heated immune serum containing the immune body but not complement)+complement (i. e., fresh serum)=no destruction of the bacteria.

(2) Bacteria+5 units immune serum+complement=complete destruction.

(3) Bacteria+1 unit immune serum+complement=no destruction.

In (3) the destruction of the bacteria is not complete, because there is not enough immune body to sensitize the bacteria to the action of the complement, or, in the terms of Ehrlich, not enough amboceptor to unite the complement to the bacterial cell.

In (2) the proper proportions of immune body and complement occur and the lytic action is complete.

In (3) there is an excess of immune body which, therefore, combines with and deviates the complement and thus renders it powerless to unite with the bacterial cell, and thereby the lytic action is prevented.

The action, therefore, while specific, is strictly quantitative, depending upon the amount, especially of immune serum, present in the mixture; that is, though immune sera protect against specific infection, they do so only in certain doses. It is easy to understand how too small an amount of immune serum will fail to protect, but difficult to understand why a large amount should fail; in other words, why an excessive amount of bacteriolytic serum should cause the bacteria to be protected rather than be destroyed.

ISOHEMOLYSINS

Isohemolysins have the property of destroying the red blood cells of the same species. They occur naturally in certain animals, principally in the horse and in man. They may also be produced experimentally in certain animals, as in goats, by the injection of the blood of other goats. There is further the possibility that *autohemolysins* may be produced which destroy the blood cells of the individual himself. These have not been produced artificially, but are said to occur in paroxysms of hemoglobinuria.

PRECIPITINS

Another class of immune bodies known as the precipitins may readily be produced in the blood serum of animals by the injection of bac-

teria or albuminous substances. The precipitating action of immune sera was discovered by R. Kraus in 1897. When the clear antiserum is added to the clear antigen in solution, the mixture of the two fluids becomes opalescent, then opaque from the formation of a precipitate, and after a time this settles to the bottom of the test tube, leaving a clear supernatant fluid. The precipitate consists of an insoluble combination of two substances, one of which is present in the antiserum, the other in the antigen. This insoluble precipitate is known as the *precipitum*. The substance in the antigen is known as the precipitable substance or *precipitinogen*, and the substance in the antiserum is called the *precipitin*. Precipitums are doubtless formed both within and without the body when proper conditions of antibody and antigen are present, without, however, always causing a visible precipitum.

The precipitins are quite analogous to the agglutinins, and from the standpoint of physical chemistry are often classified with them. It is now known that proteids do not form true solutions, but molecular or colloidal suspensions. The effect of the addition of a precipitin is to cause the agglutination of these molecules in a manner entirely analogous to the agglutination of bacilli. According to Emery, the laws which govern the action of the precipitins and agglutinins are entirely similar, and theoretically it would probably be more accurate to consider them under one head. The practical applications of the two classes of antibodies are, however, very different, and it is more convenient to treat them as separate substances.

The bacterial precipitins were those first discovered. Kraus added some typhoid serum to a filtered culture of typhoid bacilli and obtained a precipitate when the two clear solutions were brought together. The same happens with cultures of cholera, plague, and other bacteria. Certain bacteria, however, do not produce a precipitable substance. This is notably the case with diphtheria. Thus, when diphtheria antitoxin is added to diphtheria toxin, no visible reaction takes place. In this case the diphtheria antitoxin should contain the antibody or precipitinogen. The filtered broth culture is the antigen and should contain the precipitin; however, one or both of these substances must be absent, as a precipitum is not formed when they are brought together.

Tsistowitch in 1899 found that precipitins may be produced by injecting albuminous substances into suitable animals. Thus, if rabbits are injected with horse serum or with eel's blood, the blood serum of the treated rabbit will precipitate the blood serum of the horse or the eel's blood respectively. This reaction is used in forensic medicine for the recognition of blood stains, which will presently be discussed.

The chemical nature of the precipitins is not known. They come down with the globulins. In the terms of the side-chain theory they

contain two groups, one a thermostable haptophore or combining group, the other a thermolabile functioning group. Precipitins are destroyed by heat, light, moisture, and other external influences about as readily as the agglutinins. Precipitating sera should, therefore, be kept in a dry state, in a cool place, and preserved from light. A proprecipitoid zone entirely analogous to the proagglutinoid zone is observed under certain conditions. Precipitins like agglutinins act more quickly at the body temperature and require the presence of certain salts for their action. According to Friedemann, the amount of precipitum formed depends on the quantity of the salts present.

The relation of precipitins to immunity is not entirely clear. There is a strong suspicion that, like all antibodies, they play some part in the mechanism of immunity in certain infections, but just what part is obscure. It is quite evident that the presence of precipitins in the blood must have valuable protective properties against the poisons of certain infections. The immunity in this case would be due to the throwing out of solution of the poison, thus rendering it insoluble and inert.

Nuttal in his "Blood Relationship" made a very careful study of the question of specificity of the precipitins.

He showed that the reaction of the precipitins, like the reaction of other similar antibodies, is relatively specific or quantitatively specific. If the antiserum is powerful enough it will react with all the bloods of animals in the same great division of the animal kingdom. Thus, a strong antihuman serum, that is, a serum obtained by injecting human blood into rabbits, will give a precipitate when this rabbit serum and human serum are brought together; it will also react with apes, monkeys, etc., but not in such high dilutions, and a slight trace of precipitum appears after a long period even when mixed with the serum of more remote mammalia, but no precipitate occurs with the blood of birds, fishes, etc. A quite similar relationship holds with lactosera and with the precipitating sera for muscle proteids; the antisera for egg proteids are apparently less specific. Precipitins, then, are not specific as regards the animal species from which they are derived, but possess that partial specificity seen in the cytotoxins and in the group reaction of the agglutinins. According to Emery, they are specific as regards the antibodies which bring them into existence, irrespective of the source from which the antigen is derived. For medicolegal purposes the specificity of the reaction may be considered satisfactory, provided the tests are made quantitatively, in which case the reaction is both specific and delicate. In fact, the delicacy of the reaction is truly astonishing. Thus, Ascoli obtained an antiegg albumin serum which gave a precipitate with 1-1,000,000 dilution of egg albumin; and Stern an antihuman serum which reacted with serum at a

dilution of 1-50,000. While these are extreme figures, it is not unusual to obtain precipitates in dilutions of 1-5,000.

Tests for Blood.—In carrying out the precipitin tests for the recognition of blood stains, as suggested by Uhlenhuth and Wassermann, it is necessary first to obtain an antiserum. This is usually gained from rabbits, which are injected intravenously or intraperitoneally at intervals of three or four days with human serum. The human serum may readily be obtained by puncturing a vein at the bend of the elbow, or from the placenta, or from a cadaver; pleuritic or ascitic fluid may also be used. The amount injected rises from 1 to 3 or 4 c. c. in the case of intravenous injections, or twice as much or even more into the peritoneum. The course of treatment lasts three or four months. A simpler method is to give larger doses up to 10 c. c. or more intraperitoneally at intervals of a week. The intervals should not be longer than this, for danger of complicating anaphylactic reactions. The blood may be drawn from a vein or the heart of the rabbit from time to time as needed, or the animal may be chloroformed and exsanguinated through the carotid artery, or as much blood as possible may be collected from the heart.

The blood to be tested is usually in the form of a clot or stains upon linen, pistols, and other surfaces. These stains are macerated with normal saline solution or with 1 per cent. sodium hydrate. In the case of very old stains Zienka recommends the use of a strong solution of potassium cyanid which is subsequently neutralized with tartaric acid. The fluid is then examined with the microscope and tested spectroscopically to determine the presence of blood corpuscles and pigments, so as to be sure we are really dealing with blood. The solution is then filtered. In order to determine the approximate strength of the solution it is sufficient to bubble air through the fluid. A dilution of blood serum in the proportion of 1-1,000 will produce a stable foam. If a stable foam is not produced it indicates that the protein material has not actually passed into solution or is too dilute to be of service in the test. Three tests are made. In the first tube one part of the fluid under examination is mixed with two parts of the antiserum, the second contains the fluid alone, and the third antiserum plus normal saline solution. Further controls in which the antiserum is mixed with diluted serum from animals other than man may also be made. The tubes are then incubated at 37° C. and examined from time to time. A positive result is obtained if there is a precipitate in the first tube and not in the others. In case a precipitate is obtained further tests are then made with greater dilutions. With a powerful antiserum a reaction may usually be obtained in dilutions so high that evidence of the presence of proteids is barely obtainable by ordinary chemical means. The weak point in the method is that it is never possible to say exactly how much of the protein matter of the clot has been

dissolved, and thus it is not possible to obtain precise quantitative results. With an unknown blood serum, unaltered, and in the fluid state the test can be carried out with almost complete certainty, but this is rarely if ever possible in medicolegal cases.

Another test for blood has been introduced by Neisser and Sachs and based on the Gengou reaction of fixation of the complement. The test is extraordinarily sensitive. Neisser and Sachs found that one-millionth part of a cubic centimeter of human serum is readily demonstrable. The technique is complicated, and, according to Emery, it appears, moreover, that complement may be extracted in an altogether non-specific manner by substances other than the combination of antigen and antibody. Another serious objection is that a similar deviation of the complement may be brought about by means of sweat, so that if the reaction were obtained in a stain on body linen it would be of little value.

The precipitin reaction further finds practical application in determining the nature of meat, whether fresh, as in the case of beef suspected to be horse flesh, or prepared, as in sausages, etc. For these tests the antiserum is prepared by injecting rabbits with meat juices or an unheated watery extract of the meat, and the test is carried out on lines similar to those described above.

AGGLUTININS

Agglutinins were definitely described in 1896 by Gruber and Durham, and a few days later by Pfeiffer and Kolle. Shortly thereafter Widal announced the fact that the blood serum of a typhoid patient will agglutinate the typhoid bacillus in high dilutions. The phenomenon of agglutination with special reference to typhoid fever is, therefore, often called the Widal reaction or the Gruber reaction.[1]

Agglutination consists in a clumping or grouping of the bacteria into clusters, just as though they were iron filings drawn about a magnetic point. Usually they are immobilized before they are drawn together into a clump or cluster. Theobald Smith has shown that the first phenomenon, the immobilization of bacteria, may be due to a flagellar agglutinin, and that the second phenomenon, the clumping, may be due to a cellular agglutinin.

The agglutination of bacteria apparently does little harm to them other than rendering them motionless, for they are not altered in ap-

[1] The phenomenon of agglutination had been previously observed by Charrin and Roger in 1899 in the case of the *Bacillus pyocyaneus*. It was also observed by Metchnikoff in the case of the *Vibrio metchnikovi* in 1891. Similar appearance had also been seen by Issaeff in 1893.

pearance, viability, or virulence. Bacteria that have been agglutinated may again multiply and grow vigorously. Agglutination is an important source of error in counting the number of bacteria in any fluid. A cluster will develop into one colony and thereby give misleading results. The apparent diminution in the number of bacteria in freshly drawn milk, judged by the number of colonies that develop upon agar plates and known as the germicidal property of milk, is largely a phenomenon of agglutination.

Agglutination may occur quickly or slowly, depending upon the temperature, the dilution of the serum or fluid containing the agglutinin, and upon other factors; hence, it is important in reporting positive or negative tests in the diagnosis of typhoid fever, malta fever, and other infections always to state the dilution, the time, the temperature, and other conditions under which the test was made. The interpretation of the results may depend upon these factors.

Agglutination may readily be seen by the naked eye. A uniform suspension of bacteria in a test tube under the action of an agglutinin first becomes granular; the granules increase in size and flock into masses with intervening clear spaces. Then these flocculi settle to the bottom as a precipitate, leaving the supernatant fluid clear. Under the microscope the bacteria are first seen to lose their motion, then to be drawn together into irregular clumps or clusters, which increase in size. The macroscopic method is much more dependable in testing agglutinins than the microscopic method. The latter is subject to several sources of error, and the end point is not as sharply defined as in the macroscopic method.

Agglutination, like almost all chemical processes, takes place more quickly when warm than in the cold. The reaction is best at 37° C. The clumping usually takes place more slowly with the non-motile bacteria. Certain strains of some species of bacteria agglutinate more readily than others. Thus, the typhoid bacillus is usually agglutinated readily with its specific serum, but some strains are agglutinated with considerable difficulty; in general, when first isolated, they resist agglutination. This resistance or "immunity" of the microorganism usually wears off after a number of subcultures. A very interesting phenomenon in agglutination which has considerable practical importance is the so-called proagglutinoid zone; that is, bacteria sometimes will not agglutinate in a stronger dilution, whereas they agglutinate readily in a weaker. The proagglutinoid zone is occasionally found with the typhoid bacillus, but especially with the *Micrococcus melitensis*. Thus, this coccus may give no reaction in a dilution between 1-10 and 1-100, whereas it will clump strongly at 1-200.

Agglutinins are not very resistant to light, putrefactive processes, and dryness. They are not much harmed at a temperature of 55° to

56° C., but are destroyed at 65° to 70° C. They are very sensitive to acids; they are partially held back by a Pasteur-Chamberland filter; they are not dialyzable. They may be preserved for a very long time in dried serum protected from light and moisture.

The chemical composition of the agglutinins is not known. Like antitoxin and other antibodies, they come down with the globulins when precipitated with ammonium sulphate. They unite directly with the bacteria or other cells and, according to Ehrlich, contain both a haptophore and an "agglutinophore" group.

Agglutinins may readily be produced by injecting either live or dead bacterial cells into a suitable animal. The injections may be given either subcutaneously, intravenously, intraperitoneally, or the microorganisms may be rubbed upon the closely shaven skin. Agglutinins may even be produced by giving the microorganisms by the mouth. Agglutinins in highest concentration may be obtained by repeated injections, every 10 or 12 days, continued over a long period of time. In experimental work in the laboratory rabbits are suitable. Three or four injections into the ear vein of the rabbit, spaced at intervals of 8 or 10 days with cultures of cholera or typhoid, will develop agglutinins in the blood serum when diluted as high as 1 to 5,000 or 1 to 10,000. Where large amounts are needed the horse is the most suitable animal.

Agglutinins also appear spontaneously in attacks of certain infectious diseases and continue in the blood for some time after convalescence. In typhoid fever they appear about the end of the first week. They are usually weak at first, clumping the typhoid bacilli in a dilution of 1-30 in one hour at the body temperature, and increase with the progress of the disease, so that the serum may agglutinate in dilutions of 1-1,000 or more. In malta fever agglutinins appear about the fifth day of the disease and may develop in large amount. Thus, the blood serum from a case of malta fever may agglutinate the *Micrococcus melitensis* in dilutions as high as 1-500,000. The reaction of agglutination is not only practical as an aid to diagnosis of disease, but is of considerable practical use as an aid of recognition of the bacteria themselves.

The reaction of agglutination is not absolutely specific; thus, a typhoid agglutinin will occasionally clump proteus or other not very closely related microorganisms. Thus, Frost found a *Psedomonas protea* in the Potomac River water that showed quite constantly the characteristic of being agglutinated by specific typhoid immune serum. However, when animals were injected with the *Ps. protea* they developed agglutinins for this organism, but not for the *B. typhosus*. Further, there is the phenomenon of group agglutination or group reaction; that is, a typhoid serum will clump the colon bacillus, the paratyphoid,

the paracolon bacillus, and closely related organisms in the colon typhoid group. However, this occurs only in weak dilutions. The reaction is, therefore, specific in a quantitative sense. Thus, a good cholera or typhoid serum will agglutinate these organisms in dilutions of 1-1,000 and over, whereas the group reactions occur in dilutions of about 1-50 or less.

In addition to the bacteria, the red blood cells, or cells of any sort, trypanosomes and other protozoa may be agglutinated.

We have no satisfactory explanation of agglutination. Analogous phenomena occur in the study of the physical chemistry of colloidal substances. It seems that in agglutination two separate phenomena are involved: the approach of the particles, one to the other, and their adhesion subsequently. The phenomenon may be imitated by coating match sticks with soap, floating them upon the surface of water in a basin, and then adding sulphuric acid. The agglutinins affect the surface tension between the bacteria and the fluid in which they are suspended in some way, but just how is not quite clear. The agglutinins are probably formed in the lymphoid organs, red marrow, and spleen; at least, Pfeiffer and Marx found them early in these organs after injections of cholera vibrios. Metchnikoff found that the peritoneal exudate may be richer in agglutinins than the blood, and believes in that fluid they come from the leukocytes and endothelial cells.

The part played by the agglutinins in immunity is not clear. Although the bacteria are immobilized, this does not particularly favor phagocytosis. Large clusters of bacteria or agglutinated clumps of closely packed cells afford a mechanical protection against the dissolving action of the lysins.

ANAPHYLAXIS

Anaphylaxis (*ana*, against, and *phylax*, guard, or *phylaxis*, protection), also called hypersusceptibility, is a condition of unusual or exaggerated susceptibility of the organism to foreign proteins. In other words, anaphylaxis is an altered power of reaction on the part of the body to foreign proteins. The word anaphylaxis was introduced by Richet to describe a condition contrary to prophylaxis. As we now regard the phenomenon, the word is a misnomer, for we look upon the condition of hypersusceptibility as a distinct benefit and advantage to the organism; in fact, immunity against a large class of infectious diseases probably depends upon an altered power of reaction, that is, upon hypersusceptibility or anaphylaxis.

The condition of anaphylaxis may be congenital or acquired, local or general, and is specific in nature. It may be brought about by the introduction of any strange protein into the body. Hypersusceptibility to

proteins that are non-poisonous in themselves may readily be induced in certain animals. The animal may be in a condition of hypersusceptibility and immunity at the same time. The two conditions are closely interwoven. The latter is often dependent upon the former. Von Pirquet suggests the term "allergie" to indicate conditions of acquired immunity associated with anaphylaxis. Allergie, as the word indicates (*allos,* change, and *ergon,* action), is an altered power of the organism to react. When this power of reaction is increased we say the body is hypersusceptible, or in a state of anaphylaxis.

Examples of Anaphylaxis.—In the case of vaccinia, the reaction to a primary "take" appears after an incubation of four days. In a secondary vaccination the period of incubation is shortened and the clinical reaction lessened. In other words, the power of the organism to react is changed. This power of accelerated or immediate reaction protects the individual. Therefore, there is no absolute immunity in the class of diseases represented by smallpox; the prophylaxis depending upon the anaphylaxis.

The tuberculin and mallein reactions are well-known instances of anaphylaxis. These substances are not poisonous when introduced into a healthy individual, but the tuberculous individual is anaphylactic to tuberculin, and an individual suffering from glanders is in a state of hypersusceptibility to mallein.

A clinical instance of anaphylaxis is the hypersusceptibility of some individuals to pollen—hay fever.

Experimental anaphylaxis may be brought about in various ways, such as the introduction of an alien serum into the body—serum anaphylaxis.

Experimental Serum Anaphylaxis.—The essential features of experimental anaphylaxis are:

(1) The *first injection,* consisting of a bland alien protein non-poisonous in itself, which sensitizes the animal;

(2) An *interval* of about 8 to 14 days;

(3) The *second injection* of the same protein which produces a reaction known as acute anaphylactic shock.

Horse serum, when injected into normal guinea pigs, causes no symptoms. As much as 20 c. c. may be injected into the peritoneal cavity of a guinea pig without causing any apparent inconvenience to the animal. Small amounts of horse serum may even be injected directly into the brain without causing any untoward symptoms.

Very characteristic symptoms, however, are produced by horse serum when injected into a susceptible guinea pig; i. e., one that has received a prior injection of horse serum. In five or ten minutes after injection the pig becomes restless and then manifests indications of respiratory embarrassment by scratching at the mouth, coughing, and sometimes by

spasmodic, rapid, or irregular breathing; the pig becomes agitated and there is a discharge of urine and feces. This stage of exhilaration is soon followed by one of paresis or complete paralysis, with arrest of breathing. The pig is unable to stand or, if it attempts to move, falls upon its side; when taken up it is limp: spasmodic, jerky, and convulsive movements now supervene. This chain of symptoms is very characteristic, although they do not always follow in the order given. Pigs in the stage of complete paralysis may fully recover, but usually convulsions appear, and are almost invariably a forerunner of death. Symptoms appear about ten minutes after the injection has been given; occasionally in pigs not very susceptible they are delayed thirty to forty-five minutes. Pigs developing late symptoms are not very susceptible and do not die. Death usually occurs within an hour and frequently in less than thirty minutes. If the second injection be made directly into the brain or circulation, the symptoms are manifested with explosive violence, the animal frequently dying within two or three minutes.

A fall in temperature occurs which in fatal cases may be as great as 13° C. (Pfeiffer). The blood during anaphylactic shock shows a leukopenia and a diminution in complement. When the chest is opened the lungs show a striking condition resembling emphysema. They do not collapse but remain fully distended, forming a cast of the pleural cavities. The heart continues to beat long after respiration has ceased. Asphyxia, due to inspiratory immobilization of the lungs, is, therefore, probably the immediate cause of death.

Judged by the severity of the symptoms of the acute anaphylactic reaction, the guinea pig is apparently the most susceptible of animals (being 400 times more sensitive than the rabbit, according to Doerr), but probably all animals may be sensitized to a greater or less degree, although our methods of observation are still too crude to admit of any accurately graded comparison. White mice were long thought to be non-responsive on account of the absence of anaphylactic shock and death from asphyxia, so striking in the guinea pig; but Schultz and Jordan have shown that white mice do react toward horse serum with restlessness, marked irritability of the skin, passage of urine and feces, and temperature and blood pressure changes.

In dogs, according to Richet, the principal symptoms are gastro-intestinal. There is immediate vomiting, followed by tenesmus and bloody discharges from the intestines. Death is infrequent, but there may develop a condition of hemorrhagic inflammation in both the large and the small intestine which is called by Richet "chronic anaphylaxis," and by Schittenhelm and Weichardt, "enteritis anaphylactica." Another important sign is the rapid fall in blood pressure, sometimes 80-100 mm.; coagulation of the blood is delayed. Dyspnea is not marked, but,

as in other animals, there is initial restlessness and skin irritability; there may be paralysis and death.

Rabbits are apt to react to a re-injection of horse serum by edema and even necrosis at the site of injection—the "Arthus phenomenon," a local anaphylaxis. Arthus also described, in non-fatal cases in rabbits, respiratory disturbance, general prostration, fall in blood-pressure, and increased peristalsis. In cases of acute lethal anaphylaxis produced in rabbits highly sensitized by repeated minute injections, Auer describes the slow respiration, the sudden falling of the animal on its side with a short clonic convulsion, stoppage of the respiration, weak heart beat, and death within a few minutes.

The reaction to a second injection of serum has been observed, though not studied so carefully, in numerous other animals, e. g., in cows, horses, goats, sheep, and cats, in hens and pigeons, and in certain cold-blooded animals, with symptoms varying according to the species.

It is evident that no one symptom, or group of symptoms, can be taken as an adequate criterion of anaphylaxis in all cases. Different species give a widely differing picture with the same proteid agent, because the same organs are not involved to the same degree. An explanation of these differences from the physiological point of view has been given by Schultz. He has shown that serum anaphylaxis is essentially a matter of hypersensitization of smooth muscle in general. He concludes, as a result of his experiments, that, during anaphylactic shock, all smooth muscle contracts. This is fatal to the guinea pig, owing to the peculiar though normal anatomical condition of its bronchial tree: the mucosal layer of the secondary bronchi is relatively thick in comparison with the lumen, and the contraction of the smooth muscle throws it into folds which completely occlude the bronchi (Schultz and Jordan). The guinea pig dies of asphyxia, the cause of which is purely local and not in the central nervous system, as the first investigators believed. The bronchi of mice, dogs, and rabbits, however, are relatively poor in mucous membrane, which accounts for the almost complete absence of death from asphyxia. In the dog the contraction of smooth muscle sets up a vigorous intestinal peristalsis and a forced emptying of the urinary bladder; the characteristic initial rise in blood pressure may be due to constriction of the pulmonary, coronary and systemic arteries, and according to Auer, the subsequent marked fall to direct action on the heart muscle itself, particularly of the right side, causing a venous accumulation of blood, an effect typified most strikingly in the rabbit. This provides also an adequate pharmacological explanation of the action of atropin and the anesthetics in alleviating the symptoms of acute anaphylaxis.

Specificity.—The anaphylactic reaction is *specific*. Thus, a guinea pig sensitized with horse serum does not react to a subsequent injection

of egg-white, vegetable proteid, or milk. The specificity extends even further than this. In order to give rise to anaphylactic symptoms, the proteid material given at the first and second injections must be from the same species or from some closely related species. Thus a guinea pig sensitized with cow's milk will not react to a subsequent injection of woman's milk. Guinea pigs sensitized with the albumen of hen's eggs will not react to a subsequent injection of the albumen of the eggs of pigeons, but do react mildly to duck egg-white. This specificity according to species is, therefore, of the same degree as that of certain immune reactions, notably the precipitins; that is, there is a group reaction in the proteids of allied species, but no reaction between the proteids of widely different species or between proteids of widely different origin. The maximum effect at second injection is obtained by the use of the identical proteid used for sensitization. Certain sera which react interchangeably to precipitins, as, for example, human and ape, horse and ass, sheep and goat, rat and mouse, remain indistinguishable also by the anaphylactic reaction. The same specificity holds with respect to bacterial proteids: an animal sensitized with typhoid bacilli will react strongly toward paratyphoid, and somewhat toward colon bacilli, but not at all to unrelated species.

One of the remarkable facts in relation to the specificity of anaphylaxis is that guinea pigs may be in a condition of anaphylaxis to three proteid substances at the same time; for instance, a guinea pig may be sensitized with egg-white, milk, and horse serum, and subsequently react separately to a second injection of each one of these substances. The guinea pig may be sensitized by giving these strange proteids either at the same time or different times, in the same place or in different places, or by injecting them separately or mixed. The guinea pig differentiates each anaphylactogenic proteid in a perfectly distinct and separate manner. The animal is susceptible to the second injection of each one of the three substances in the same sense that it is susceptible to three separate infectious diseases.

That there may be exceptions to the rule of species-specificity is shown in the case of the crystalline lens. A guinea pig sensitized to the lens-extract of one species of animal will react to the lens-extract of widely different species, or even of its own species, but not to other tissues (Andrejew). Here, too, there is an exact parallel in the precipitin reaction which fails to distinguish the lens of one species from that of another (Uhlenhuth). This is an example of organ-specificity. In the vegetable world Osborne has shown that, whereas preparations of globulins from hemp, flax, and squash do not react with each other, gliadin from rye reacts strongly with gliadin from wheat, a result in accord with the fact that by chemical and physical means no differences have

been detected which were sufficient to indicate that these gliadins were different substances.

It is probable that only proteids which have a complete or partial chemical identity of structure will react with each other. Differences too small to be detected by analytic means at our disposal may yet prevent any tendency toward interaction, and the anaphylactic phenomenon may thus be used to determine the finer relationships of proteids. It is evident from these facts, as Osborne concludes, that structural differences exist between very similar proteids of different origin, and that chemically identical proteids apparently do not occur in animals and plants of different species unless they are biologically very closely related.

Sensitization by Feeding.—Guinea pigs may be sensitized by feeding them meat or serum. The fact that guinea pigs may be rendered susceptible by the feeding of strange protein matter opens an interesting question as to whether sensitive guinea pigs may also be poisoned by feeding with the same protein given after a proper interval of time. If man can be sensitized in a similar way by the eating of certain protein substances, this may throw light on those interesting and obscure cases in which the eating of fish, sea food, or other articles of diet sometimes cause sudden and often serious symptoms resembling those of anaphylaxis in all essential respects.

Maternal Transmission.—It has been found that hypersusceptibility to the toxic action of horse serum is transmitted from the mother guinea pig to her young. This function is solely maternal; the male takes no part whatever in the transmission of these acquired properties. Whether this maternal transmission is hereditary or congenital cannot be definitely stated.

There are certain analogies between the action of tuberculosis and horse serum. Both produce hypersensitiveness and also a certain degree of immunity. Now that it has been proved that hypersensitiveness or anaphylactic action may be transmitted in guinea pigs, may it not throw light upon the fact that tuberculosis "runs in families"? While there are several recorded instances demonstrating that immunity to certain infectious diseases may be transmitted from a mother to her young, this is, so far as is known, the only recorded instance in which hypersensitiveness or a tendency to a disease has been experimentally shown to be transmitted from a mother to her young.

Serum Anaphylaxis in Man, or Serum Sickness.—Serum anaphylaxis in man is met with most frequently following the use of antitoxic sera, and has been carefully described by v. Pirquet and Schick (1905).[1] After an injection of serum (usually in from eight to twelve days) there is apt to be a febrile reaction, now generally known as "serum-sickness," or serum disease. The common symptoms are local redness, itching and

[1] "Serum Krankheit," Wien, 1905.

pain at the point of injection, swelling of the lymph nodes, fever, and a general urticaria lasting from two to six days. In more severe cases there is malaise, albuminuria, pronounced joint pains and even effusions, swelling of the mucous membranes, hoarseness and cough, nausea and vomiting, vertigo, and remarkable skin manifestations varying from hyperemias and erythemas to efflorescences resembling measles or scarlatina, and other vasomotor disturbances.

Rarely there may be subnormal temperature, a weak and rapid pulse, a catarrhal or hemorrhagic enteritis and extreme weakness approaching collapse. These results are independent of the antitoxic qualities of the serum, for Johannessen obtained the same symptoms by introducing normal horse serum into the bodies of perfectly healthy human beings. Indeed, the very earliest animal experiments were particularly concerned in determining whether the antitoxin played any part in the phenomenon, and it was soon conclusively eliminated as a factor.

Both the incidence and the severity of serum sickness are proportional to the amount injected up to a certain point, but the acute (sometimes fatal) reaction in man is more dependent upon the hypersusceptibility of the individual than upon the amount of serum injected. If the serum is "concentrated" (i. e., serum-globulin), the reactions are correspondingly lessened because smaller quantities of the foreign proteid are injected, the albumens and certain other proteids having been eliminated by the partial purification.

The peculiarity of serum sickness in man is that it may follow the first injection of a foreign serum, though only after a definite incubation period corresponding to the time required to sensitize an experimental animal. There is no proof that other animals do not develop a reaction to the first dose which never rises to the threshold of clinical observation; in fact, Ehrlich, Francione, and others have observed a temporary diminution of complement in the blood of guinea pigs 10-12 days after the first injection.

Besides the typical serum sickness, there has been reported since the introduction of serum therapy a certain small number of unforeseen and fatal catastrophes attending the injection of serum into human beings. The following case published by H. F. Gillette will serve to illustrate them all:

"The patient was a man of 52, a subject of asthma. He asked me to administer diphtheria antitoxin to him, hoping it might cure his asthma. I administered 2,000 units under the left scapula with the usual precautions. He had about completed dressing when he said he had a pricking sensation in the neck and chest; soon he sat down and said he could not breathe, nor did he breathe again. . . . His pulse at the wrist remained regular and full for some time after respiration ceased. He had a mild degree of cyanosis and edema of the face.

He died in tonic spasms ten minutes after injection. Autopsy revealed no palpable cause of death."

The same author collected 28 cases of collapse or death after serum injection, of which 15 died. There was a common history of previous asthmatic trouble in all but five of the 28, and all, after injection, showed common symptoms of sudden intense dyspnea, a sense of overwhelming anxiety, edema and cyanosis of the face, a sudden massive urticaria, tonic muscular spasms and continued beating of the heart long after the ceasing of respiration. Rosenau and Anderson collected 19 cases and were able to examine the serum used in two of them. It was found to be no more toxic to sensitized guinea pigs than normal horse serum. These cases of severe systemic shock seem susceptible of no other explanation than that the unfortunate individuals had been in some manner, at a previous time, sensitized to horse proteid. They present a picture which is almost the counterpart of typical anaphylactic shock in guinea pigs, and the most striking thing about them is that practically all give a history of respiratory trouble in the past, especially horse-asthma. Schultz and Jordan suggest that these occasional cases of sudden death in man may perhaps be due to an abnormal development of the mucous membrane and smooth muscle of the bronchi (as in asthmatics), and that the smooth muscle, being hypersusceptible, produces asphyxia by sudden contraction. Rosenau and Amoss[1] have recently indicated a possible explanation of the way in which such persons may become sensitized. They have proved that a proteid material is given off in the expired breath of human beings. There is some reason to suppose that the proteid given off by one animal may be absorbed by individuals of different species by way of the lungs. One thing is clear, that these immediate and sometimes fatal reactions are not dependent upon any peculiar property in the serum, but to an altered power of reaction of the individual to the foreign proteid injected. The anaphylactic reactions following the injection of serum in man may be summed up briefly as follows:

Reactions following first injection:

(a) "Serum sickness," incubation 8-12 days (common).

(b) Acute anaphylactic shock, with collapse or death (rare).

Reactions following second injection:

(a) Interval between injections less than 8 days, no reaction.

(b) Interval 12-40 days, immediate reaction.

(c) Interval 15 days-6 mos., either immediate or accelerated reaction, or both.

(d) Interval over 6 mos., accelerated reaction.

[1] Rosenau, M. J., & Amoss, H. L.: *Jour. of Med. Res.*, Sept., 1911, XXV, 1, pp. 35-84.

The above table represents the usual course of events, but exceptions may occur, and the time intervals are only approximate. Sometimes the reactions do not appear until the third, fourth, or some subsequent injection.

Two precautions are suggested in serum therapy:

(1) Except in urgent cases, avoid injecting horse serum into individuals known to be asthmatic, especially those whose symptoms are brought on by being around horses.

(2) If hypersensitiveness is suspected, give at first a very small portion of the dose, following it in an hour or so with the rest, injecting it exceedingly slowly and avoiding direct injection into the circulation.

Hypersusceptibility and Immunity Produced by Bacterial Proteins.—The problem of hypersusceptibility has an important bearing on the question of immunity, and hence the opinion has been expressed that "resistance to disease may largely be gained through a process of hypersusceptibility. Whether this increased susceptibility is an essential element or only one stage in the process of resistance to disease must now engage our attention." We cannot escape the conviction that this phenomenon of hypersusceptibility has an important bearing on the prevention and cure of certain infectious processes.

Hypersusceptibility may easily be induced in guinea pigs with protein extracts obtained from the bacterial cell. The first injection of most of the extract seems comparatively harmless to the animal. A second injection of the same extract shows, however, that profound physiologic changes have taken place. A definite period must elapse between the first and the second injection. The symptoms presented by the guinea pigs as a result of the second injection resemble those caused by horse serum. The phenomenon induced by a second injection is followed (in certain cases) by an immunity to the corresponding infection.

These results strengthen the belief that the phenomenon of hypersusceptibility has a practical significance in the prevention and cure of certain infectious processes. It also gives a possible explanation of the period of incubation of some of the communicable diseases. Is it a coincidence that the period of incubation of a number of infectious diseases is about ten to fourteen days, which corresponds significantly with the time required to sensitize animals with a strange protein?

In certain infectious diseases with short periods of incubation, such as pneumonia, the crisis which commonly appears about the tenth day may find a somewhat similar explanation. It is evident that disease processes produced by soluble toxins, such as diphtheria and tetanus, do not belong to the category now under consideration.

Relation of Anaphylaxis to Protein Metabolism.—The whole problem of protein metabolism seems to be an adjustment in the sense of a

defense. The power to assimilate and use foreign proteins is not achieved without a certain amount of violence to the body. The relation between the fundamental facts of protein metabolism and immunity to certain diseases becomes clearer in the light of observations upon anaphylaxis. A deeper insight into these problems will throw light on the fundamental processes concerned in both protein metabolism and immunity.

Relation of Anaphylaxis to Endotoxins.—The fact that the great majority of bacteria do not produce soluble poisons, such as diphtheria and tetanus, has led to the belief that in such cases we are dealing with an "endotoxin." The endotoxin has long been regarded as a poisonous substance so intimately associated with the cell that it is not released until the microbic cell is broken up in the body. The inability to demonstrate many of these endotoxins has cast a doubt on their existence and increased the mystery of their action. It now seems probable that the studies on anaphylaxis may throw light upon this question.

When bacteria grow in the body they are dissolved by lytic agencies and the foreign protein in the individual germ cells may sensitize the body and afterward poison it. The bacterial proteins may not be poisonous in themselves in the sense of an "endotoxin." We have, in fact, shown that protein extracts of bacterial cells at the first injection may produce characteristic symptoms, and this reaction may be followed by an immunity to the corresponding infection.

The Relation of Anaphylaxis to Tuberculosis.—The tuberculin reaction is one of the best known instances of anaphylaxis. Following a local infection with the tubercle bacillus the tissues generally become hypersusceptible to tuberculin. It has been shown that a local hypersusceptibility may be produced by the direct application of tuberculin to certain tissues (conjunctiva). The same has been demonstrated for the skin, and is probably true of other tissues. This hypersusceptibility of the tissues immediately surrounding a tuberculous focus helps to encapsulate and limit the process. Should a tubercle bacillus lodge in or on a tissue in a state of tuberculin anaphylaxis, the result is that all of nature's protecting agencies are quickly concentrated on the point where most needed. We conceive that this active power of reacting quickly is not only an important factor in individual prophylaxis against tuberculosis, but is an important agency by which the spread of the disease after it has obtained a lodgment in the body is prevented.

The normal individual does not react to tuberculin. The tuberculous individual reacts promptly, except in the final stage of the disease. The difference between the normal individual and the individual in the final stage of tuberculosis is that the former has not had his anaphylactic powers developed, while the latter has had them developed and exhausted. A tuberculous individual in whom the specific power

of hypersusceptibility to the poisons of the tubercle bacillus is broken down presents little or no resistance against the advance of the infection.

We may adduce a practical lesson from this. When tuberculin is used in large or too oft-repeated doses there is a tendency to break down or to exhaust the useful and beneficial hypersusceptible state of the tissues. In accordance with this line of reasoning, therefore, tuberculin would be of benefit in tuberculosis only when used in such a way as to develop and not diminish the power of anaphylaxis of the tissues. This explanation has been borne out in the use of tuberculin.

Relation of Anaphylaxis to Vaccination.—When the virus of cowpox is introduced into the skin we implant a colony of microorganisms. They grow day by day, and on the eighth day there is an enormous number of them. The contents of the vesicle will start new colonies on thousands of other arms, but now the antibodies appear and the colony is attacked and digested, and toxic bodies are formed. This is diffused in the neighborhood and we get an intense local inflammation called the areola. Some of the toxic bodies enter the circulation and cause fever, but the microorganisms are killed and we can no longer vaccinate with the contents of the now yellow pustule; two or three days more, the struggle is over, but the antibodies remain a long time. Let us now revaccinate, and a different series of events takes place, for in the meantime the body has become educated and instead of waiting some days before attacking the colony of microorganisms in the skin, starts the attack at once. In other words, there is an immediate reaction—a changed power of reaction or anaphylaxis. In brief, the first vaccination has sensitized the tissues, so that they respond at once upon the second vaccination.

The invading microorganisms, attacked at once, are soon digested—they are given no chance to multiply, and little toxin is formed. This attractive explanation of the immunity to smallpox or cowpox, developed by von Pirquet, shows that the prophylaxis depends upon the anaphylaxis.

Other Practical Relations of Anaphylaxis.—In addition to hay fever, already mentioned, there are a number of other conditions which find their best explanation as examples of local anaphylaxis. This includes many of the urticarias and sudden vasomotor disturbances of the mucous membranes; various forms of asthma are also associated with hypersusceptibility to foreign substances. Idiosyncrasies with regard to articles of diet belong to the same category. Some persons are sensitized to pork, others to eggs, and sensitization to sea-food is common. Other conditions which have been explained in whole or part on the theory of anaphylaxis are puerperal eclampsia, sympathetic ophthalmia, the onset of labor, the crisis in pneumonia, the spasmophilic diathesis, the symptoms attendant on the rupture of the cysts in echino-

coccus disease, etc. The anaphylactic reaction is also used in diagnosis, and in forensic medicine in the identification of blood stains, and, finally, may be used as a scientific instrument for the detection of minute amounts of protein.

References.—Many of the statements contained in this chapter have been taken from Emery's splendid book upon "Immunity and Specific Therapy," which is recommended to the reader who desires a more extended review upon the subject. Kolle and Wassermann's "Handbuch der Mikroorganismen" has also been consulted, as well as Kraus and Levaditi's "Handbuch der Technik und Methodik der Immunitätsforschung." These volumes also contain selected bibliographies.

The current literature upon immunity will be found in the *Zeitschrift für Immunitätsforschungen.*

For those who desire to dip deeper into the subject the original reference to many of the fundamental studies will be found in "Collected Studies on Immunity" by Ehrlich, translated by Bolduan; "Studies on Immunity" by Bordet, translated by Gay; "Studies in Immunization" by Wright, and "L'Immunité dans les Maladies Infectieuses" by Metchnikoff, translated by Binnie.

CHAPTER II

HEREDITY AND EUGENICS

Heredity may be defined as the genetic relation between successive generations. It is a condition of all organic evolution. Castle defines heredity as organic resemblance based on descent.

It is now perfectly evident that heredity is one of the fundamental factors in preventive medicine—which, after all, is the true sociology. It is well known to students of biology that education and environment have but a limited power to influence imperfect human protoplasm.

One of the best protections we have against diseases of body and mind is that which is inherited from our forebears. The whole problem of improving the human stock, not only from the medical view, but from the broader sociological standpoint, is based upon the breeding of the fit and elimination of the unfit. The science of eugenics (normal genesis), therefore, assumes especial importance in preventive medicine. The physician, as well as the sanitarian, stands impotent before many deplorable conditions both in the individual and in society at large, which are inherited from our ancestors and are, therefore, incurable—but largely preventable. We are interested in educating the present generation to the facts of eugenics so that future generations may have that best of all birthrights—good human protoplasm.

The discoveries of Mendel have made it quite clear how certain characters are inherited, why certain characters skip a generation and reappear in the grandchildren, and why it is that certain defects are carried from generation to generation through many centuries.[1] The defects transmitted hereditarily are not all of equal practical importance. Thus, it makes comparatively little difference to the individual if he has a supernumerary spleen, an extra finger, or an anatomical anomaly of the liver. The defects which are of especial importance both to the individual and to succeeding generations are the defects

[1] Mendel's work has not only made it possible for us to predict with precision whether good or bad traits will or will not appear in the future offspring, but also to foretell with mathematical precision in what proportion certain characters will appear and reappear.

of organization of the nervous system. These comprise the class known as defectives. A slight defect in the structure of the brain which would be unnoticed in the lung, bone, or musculature may render the individual vicious instead of useful. The principal factors which are believed to start a line of defectives are inbreeding, syphilis, and alcohol; also nervous or physical diseases, mental or nervous exhaustion, and excesses and poisons of all kinds.[1]

The defective individual is very easily recognized when the condition is well marked. The mental abnormality is usually accompanied by prominent physical defects known as the stigmata of degeneration (Lombroso and Weismann). An unfortunate side to this problem is that degenerates and defectives generally are not only irresponsible morally, but are very prolific. They lack self-control and have abnormal sexual appetites. Defectives beget defectives, and thus insanity, nervous diseases, moral and physical degeneracy are propagated. The typical degenerate is of poor bodily development; the brain is smaller than normal, with convolutions less abundant, and less fully formed. He has a degraded physiognomy, lacks capacity for sustained attention or for prolonged thought, is cunning rather than intelligent, deficient in moral sense—in all points resembling the stigmata of the lower, less developed races of our species. The whole gives the impression of a reversion to a lower type.

Prevention of Propagation of Defectives.—Four methods have been proposed to prevent the propagation of defectives: (1) education; (2) legislation; (3) segregation; (4) surgery.

Education.—Education directed toward the defective is a failure, for he is incapable of profiting by the lessons. The education of the better class of the community is indirectly helpful in calling attention to the situation as being largely preventable, and to the necessity and means for controlling it.

Restrictive Legislation.—Restrictive legislation is a praiseworthy effort, but has signally failed as a preventive measure, for the evident reason that it only adds illegitimacy to degeneracy, and thus the children enter on life's battle doubly handicapped. Minnesota has a law providing that within the bounds of the state no marriage shall be permitted, either party to which is epileptic, imbecile, feeble-minded, or afflicted with insanity, unless the woman be over forty-five. Michigan, Delaware, Connecticut, Indiana, New Jersey, and North Dakota have also passed laws for the purpose of preventing marriage among defectives.

Segregation.—Segregation would be an ideal and humane method

[1] The real cause or method of origin of defective characters that are transmitted hereditarily is no better understood than the origin of "sports" or mutations.

of eliminating those who are incapable of having normal offspring. The segregation of all degenerates and defectives would be an enormous and impractical task. Further, the great difficulty is to detect the unfit individual who starts a strain of defectives and degenerates. It is evidently a hopeless task to know where to draw the line between the fit and the unfit, so that for the present we must be satisfied to enforce restrictive measures upon only those who are evident and well-marked examples. Insane asylums, homes for epileptics, reformatory schools, as well as special hospitals and institutions for advanced cases must not be regarded as preventive measures in the true sense, for such segregation provides care and comfort as a terminal measure; that is, it is usually a last resort. Frequently defectives propagate their kind before and sometimes after they are segregated.

Surgery.—Surgery has been proposed as a means of controlling the propagation of defectives. This is done either by severing the *vas deferens* or the Fallopian tube. At the Indiana Reformatory Dr. Sharp carries out the law [1] of that state providing for the sterilization of defectives. The operation of vasectomy consists of ligation and resection of a small portion of the vas deferens. The operation is very simple and easy to perform. It may be done without an anesthetic, either local or general. As performed by Dr. Sharp it requires about three minutes, and the subject returns to his work immediately, suffering no inconvenience and in no way hampered in his pursuit of life, liberty, and happiness, but is effectively sterilized. In 456 cases Dr. Sharp has had no unfavorable symptoms. The operation is performed as follows: After cleansing the scrotum with soap and water, followed by alcohol, the spermatic cord is grasped between the thumb and index finger of the left hand. The vas deferens is detected, firmly held and fixed with a pair of bullet forceps. It is then exposed by a small incision and drawn through the scrotum wound by means of a tenaculum. It is stripped of all membranes and the accompanying artery ligated above and severed, care being taken to cut away any por-

[1] The Indiana law reads as follows:

Whereas, Heredity plays a most important part in the transmission of crime, idiocy, and imbecility;

Therefore, Be it enacted by the General Assembly of the State of Indiana, That on and after the passage of this act it shall be compulsory for each and every institution in the State, entrusted with the care of confirmed criminals, idiots, rapists, and imbeciles, to appoint upon its staff, in addition to the regular institutional physician, two (2) skilled surgeons of recognized ability, whose duty it shall be, in conjunction with the chief physician of the institution, to examine the mental and physical condition of such inmates as are recommended by the institutional physician and board of managers. If, in the judgment of this committee of experts and the board of managers, procreation is inadvisable and there is no probability of improvement of the mental and physical condition of the inmate, it shall be lawful for the surgeons to perform such operation for the prevention of procreation as shall be decided safest and most effective. But this operation shall not be performed except in cases that have been pronounced unimprovable. . . .

tion of the vas deferens that may have been damaged in the manipulation. This is done in order that the end next to the testicle may not become closed. It is very important that the testicular end shall remain open, in order that the secretion of the testicle may be emptied around the vessels of the pampiniform plexus and there be absorbed, for it is through this process that the body receives the tonic effect of the internal secretion. Further, if the testicular end of the vas deferens is closed, there is likely to be cystic degeneration of the testicle. The retraction of the muscle closes the skin wound and no stitch, collodion, or adhesive plaster is needed. There is no diminution of the sexual power or pleasure. The discharge at the orgasm is but slightly decreased.

The operation in the female is more difficult, but if carefully done no more hazardous. The Fallopian tubes are reached through a median incision and ligated near the uterus and severed beyond the ligature.

Opinions vary greatly concerning the proper use of sterilizing criminals, insane, degenerates, and defectives generally. There is no doubt concerning its effectiveness.

Sterilization is a measure which contains great potential possibilities for abuse and injustice. It probably will never receive general acceptance on account of the difficulty of determining upon whom the operation shall be done. Even in perfectly clear cases, such as the insane, the epileptic, or the high grade degenerate, the harm has often been done before the operation is decided upon.

Statistics of Defectives.—The large number of defectives and unfit in our country may be gleaned from the following figures.

The last census report for the United States gives data relative to the dependents and defectives in institutions; the number not in institutions can only be guessed at. Kellicott gives the following approximate numbers in our country to-day:

Insane and feeble-minded, at least	200,000
Blind	100,000
Deaf and dumb	100,000
Paupers in institutions	80,000
Prisoners	100,000
Juvenile delinquents in institutions	23,000

The number of persons cared for in hospitals, dispensaries, "homes" of various kinds in the year 1904 was in excess of two million.

We have to support about half a million insane, feeble-minded, epileptic, blind, and deaf; 80,000 prisoners, and 100,000 paupers, at a cost of $100,000,000 per year. A new plague affecting 4 per cent. of the population and costing this vast treasure would instantly attract uni-

versal attention. We have become so used to crime, disease, and degeneracy that we take them as necessary evils. "That many of them were so in the world's ignorance is granted; that they must remain so is denied."

Statistical studies seem to indicate a rapid (at least an unnecessary) increase of the unfit, defective, insane, criminal, and, on the other hand, a slow increase, or even a decrease (?), of the fit, normal, or gifted stocks. It is plain to the student of eugenics how such conditions account for the rise and fall of nations.

The United States census of 1880 reported 40,942 insane in hospitals and 51,017 not in hospitals; a total of 91,959 known insane. In 1903 it was estimated that there was a total of 180,000 in the United States. Thus, the ratio of known insane in the total population was 225 per 100,000 in 1903, as compared with 183 per 100,000 in 1880. These figures must not be taken as an index of the increase of insanity in the population at large—for institutional care has been growing much more popular during the past decade, especially since more humane methods have been adopted. Further, the classification of insanity now includes many cases that were formerly little noticed.[1]

The comparatively large and increasing numbers of defectives and weaklings among the civilized races compared with wild animals may be accounted for by the fact that atavism and reversion are more frequently met with in artificially cultivated strains, such as civilized man; and the further fact that our charitable and philanthropic efforts foster and even favor the unfit.

Degenerate Families.—A careful study has been made of the records of several families in which the mating of unfit individuals has begotten a swarm of unfit descendants.

[1] A special census of the insane confined in institutions was taken by the Bureau of the Census in 1910, and it was found that 187,454 patients were confined in hospitals for the insane in the continental United States.

While the population of the United States increased about 11 per cent. in the interval between 1904 and 1910, the population in insane asylums increased about 25 per cent. The number of insane in asylums per 100,000 population increased from 186.2 in 1904 to 203.8 in 1910. The number of persons annually committed to hospitals for the insane per 100,000 population increased from 61.5 in 1904 to 65.9 in 1910. If these ratios are accepted as representing insanity rates, it would appear that the number of persons becoming insane, in a community comprising 100,000 persons, was greater by 4.4 in 1910 than it was in 1904. It must be remembered, however, that these figures include only the insane who are committed to hospitals. As to the number of cases of insanity not resulting in commitments to hospitals the census has no data. It is entirely possible that the increase in the number of commitments per 100,000 population is not due to any considerable degree to an increased prevalence of insanity, but simply to the extension of this method of caring for the insane. It is a change which might result from an increase in the number of institutions of this class and from the increasing disposition on the part of the public to resort to such institutions. In this connection it may be noted that the number of institutions for the insane reported by the census increased from 328 in 1904 to 372 in 1910, an increase of about 13 per cent. The average number of inmates per institution increased from 458 in 1904 to 504 in 1910.

One of the best known families of this type is the so-called Jukes family of New York State investigated by Dugdale. This family is traced from the five daughters of a lazy and irresponsible fisherman born in 1720. In five generations the descendants of Jukes numbered about 1,200 persons, including nearly 200 who married into it. The histories of 540 of these are well known, and about 500 more are partly known. Some 300 died in infancy. Of the remaining 900, 310 were professional paupers living in almshouses (a total of 2,300 years); 440 were physically wrecked by their own diseased wickedness; more than half of the women were prostitutes; 130 were convicted criminals; 60 were habitual thieves; 7 were murderers. Not one had even a common school education; only 20 learned a trade, and 10 of these learned it in State's prison. The descendants of Jukes in five generations have cost New York State over one million and a quarter dollars, and the cost is still going on.

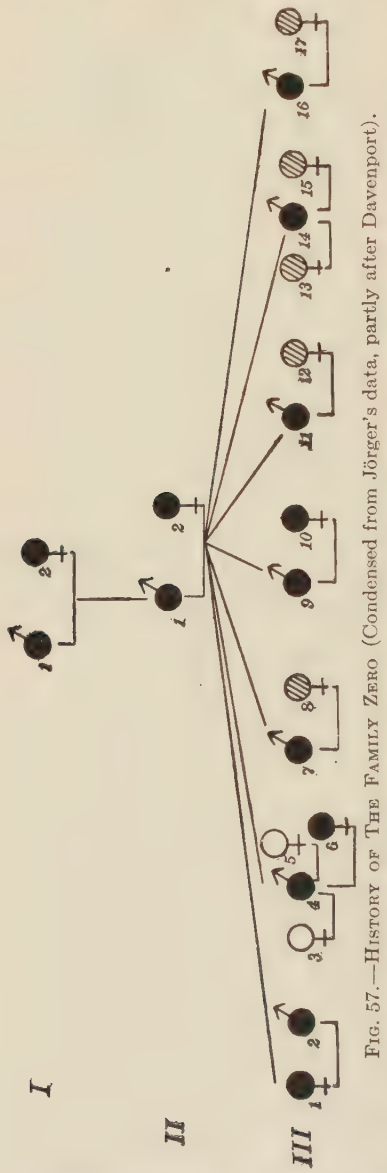


Fig. 57.—History of The Family Zero (Condensed from Jörger's data, partly after Davenport).

Probably the most complete family history of this kind ever worked out is that of the "Familie Zero," a Swiss family whose pedigree has been studied by Jörger. In the seventeenth century this family divided into three lines. Two of these have ever since remained valued and highly respected families, while the third has descended to the depths. This third line was established by a man who was himself the result of two generations of intermarriage, the second tainted with insanity. He was of a roving disposition, and in the Valla Fontana found an Italian vagrant wife of vicious character. Their son inherited fully the parental traits and himself married a member of a German vagabond family—Marcus. This marriage sealed the fate of their hundreds of descendants. The pair had seven children, all characterized by vagabondage, thievery, drunkenness, mental and physical

defects, and immorality (Kellicott). How much of this is due to heredity and how much to environment will be discussed presently.

Another interesting example of the same type has been described by Poellmann. This family was established by two daughters of a woman drunkard who in five or six generations produced, all told, 834 descendants. The histories of 709 of these are known. Of the 709 107 were of illegitimate birth, 64 were inmates of almshouses, 162 were professional beggars, 164 were prostitutes, and 17 procurers, 76 had served sentences in prison, aggregating 116 years, 7 were condemned for murder.

Dr. Henry H. Goddard[1] has investigated and compiled the results of his work on the heredity of a most remarkable family, the Kallikak family. During the Revolutionary days, the first Martin Kallikak (the name is fictitious), descended from a long line of good English ancestry, took advantage of a feeble-minded girl. The result of their indulgence was a feeble-minded son. This son married a normal woman. They in turn produced five feeble-minded and two normal children. Practically all of the descendants of these defectives have been traced, as well as those of the two normals.

From both normal and defective descendants of this union came a long line of defective stock. There were 480 in all. Of these thirty-six were illegitimate, thirty-three sexually immoral, twenty-four confirmed alcoholics, and three epileptics. Eighty-two died in infancy, three were criminal, eight kept houses of ill fame, and 143 were distinctly feeble-minded. Only forty-six were found who were apparently normal. The rest are unknown or doubtful. But the scion of the good family who started this long line of delinquent and defective progeny is also responsible for a strain of an entirely different character. After the Revolutionary War was over, he married a Quaker girl of good ancestry and settled down to live a respectable life after the traditions of his forefathers. From this legal union with a normal woman there have been 496 descendants. All of these except two have been of normal mentality. The exceptions were cases of insanity, presumably inherited through marriage with an outside strain in which there was a constitutional psychopathic tendency. In all the 496 there is not an instance of feeble-mindedness. The offspring descended from this side of the house have universally occupied positions in the upper walks of life. They have never been criminals or ne'er-do-wells. On the other hand, there has not been a single instance of exceptional ability among the descendants of the first Martin Kallikak and the feeble-minded girl. Most of these descendants have failed to rise above the dead level of

[1] "The Kallikak Family, a Study in the Heredity of Feeble-mindedness," New York, Macmillan Company, 1912.

mediocrity; indeed, most of them have fallen far below even this minimum standard.

The fact that the descendants of both the normal and the feeble-minded mother have been traced and studied in every conceivable environment, and that the respective strains have always been true to type, tends to confirm the belief that heredity has been the determining factor in the formation of their respective characters. In the cities the descendants of the legal marriage with the normal woman are physicians, lawyers and prominent business men, while the descendants of the feeble-minded mother are almost invariably found in the slums. In the rural districts the descendants of the normal mother and her consort are wealthy and influential farmers, while the others never rise above the rank of farm laborers and shiftless men and women who are unable to subsist without the aid of charity. Many representatives of the defective branch are inmates of almshouses, while there are no paupers at all among the normal descendants.

In many ways this study of Goddard's far outweighs in importance the famous comparison by Dr. Winship of the Jukes and Edwards families. In that case the simple fact was demonstrated that a good family like that of the illustrious Jonathan Edwards had given rise to innumerable examples of the highest intellectual and moral worth, whereas the criminal Jukes for seven generations contributed nothing to the common good and cost the state of New York large sums of money. But the Jukes family and the Edwards family had no ancestor in common. Their environment was totally different and they lived in entirely separate communities. Although from sociologic and economic points of view the history of the Jukes family and its comparison with that of the family of Jonathan Edwards has great value, it is of but scant scientific importance as compared with that of the Kallikak family, for here a natural object-lesson in eugenics shows unmistakably the manner in which after-coming generations from a given mating receive the characteristics of the dominant strain, which in the elder (illegitimate) Kallikak line was the inferior strain, with only a debased and enfeebled heritage to hand on.[1]

In contrast to these we have the descendants of the families of Wedgwood, Darwin, and Galton, the Edwards family and the Ward family. These three noted families contained a large number of statesmen, jurists, professors, physicians, officers in the army and navy, prominent authors and writers, and occasionally men and women of genius. They show a long line of usefulness in every department of social progress, and not one of them ever has been convicted of a crime.

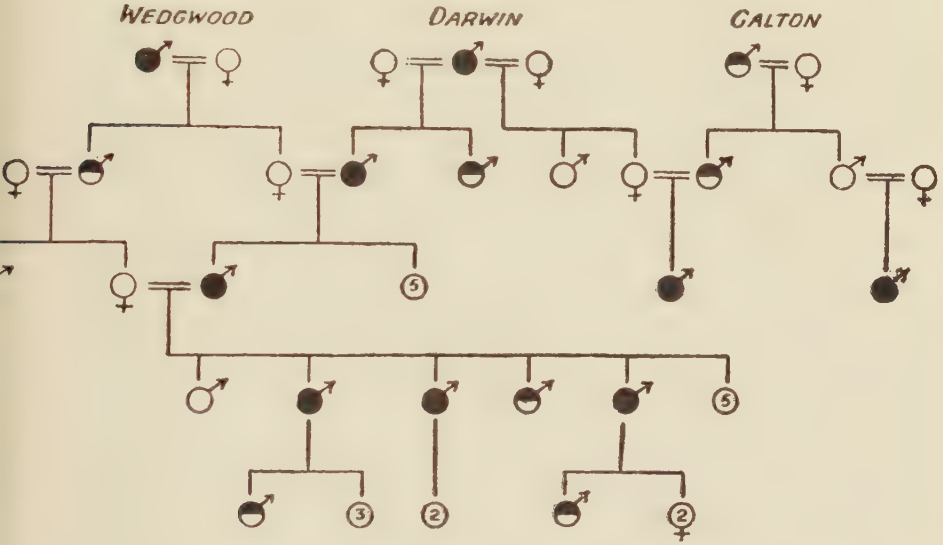
How much of this is due to heredity and how much to environment are debatable questions. Students of biology are convinced that heredity

[1] *J. A. M. A.*, Oct. 26, 1912, LIX, 17, p. 1545.

plays the major rôle in the lives of the individuals in the above-mentioned families. In how far such extreme instances as those given above represent the rule or exceptions will require much additional data and long years of study to determine.

EUGENICS

The science of eugenics has been defined as "the science of being well born." According to Galton, "eugenics is the study of the agen-



shows a man of scientific ability; shows a man of scientific ability, who is also a Fellow of the Royal Society; (5) shows five other children, and so on.

FIG. 58.—HISTORY (CONDENSED AND INCOMPLETE) OF THREE MARKEDLY ABLE FAMILIES (After Whentham) (Kellicott).

cies under social control that may improve or impair the racial qualities of future generations either physically or mentally."

The aim of eugenics is to increase the number of best specimens in each class; that done, leave them to work out their common civilization in their own way. It also aims to leave a good heritage to the next generation and to repress the propagation of the vicious and defective classes.

The success of eugenics depends almost entirely upon our knowledge of heredity and sociology. Therefore, the fundamental principles of heredity should be familiar to all students of preventive medicine.

The present movement started in 1865 when Francis Galton showed that mental qualities are inherited, just as are physical qualities, and

pointed out that this opened a way to an improvement of the race in all respects. The student should read Galton's work on "Hereditary Genius," published in 1869, when he again emphasized definitely the possibility and desirability of improving the natural qualities of the human race. The word "eugenics" was coined in 1883 in his "Inquiries Into the Human Faculty."

There is no doubt concerning the desirability of breeding better human stock, but how this may be accomplished practically is a difficult question. The program of the eugenist is perplexing and complicated. To follow the theoretical extremists would require a social revolution—a change from the present method of haphazard mating. The threshold of the subject has scarcely been passed, and we must bear in mind that some of the striking men of genius from whom the world has greatly profited have been individuals whom the student of genetics would regard as degenerates or defectives. Eugenics does not mean free love, nor does the eugenist recommend Burbanking the human race to produce great physical strength, beauty, endurance, mental or moral power. One point only in the program is perfectly clear, and that is that a check should be placed upon the propagation of the crop of defectives by means already pointed out.

The known facts of heredity and the study of eugenics make us examine more critically some of the directions which preventive medicine, including philanthropy and social uplift, has taken. We must now ask ourselves the question whether it would not be better for the future generations if we helped the fit rather than the weakling and the unfit. These are problems raised by Galton, who questions whether some of our charitable efforts are well balanced and well directed.

The importance of eugenics in medicine is not new. For a while, however, the medical sciences lost sight of heredity, owing to the ultra-materialistic view of disease which became the vogue as a result of the germ theory. A neglect of the personal element in medicine and a wholly impersonal hygiene were laid down as universally applicable. Davenport states: "It has forgotten the fundamental fact that all men are created *bound* by their protoplasmic make-up and *unequal* in their powers and responsibilities."

It is evidently now of great importance to collect a large number of pedigrees, in which the data shall be stated with scientific exactness and in minute detail. Such a mass of facts may then be studied in the light of our present knowledge in order to determine in how far the laws of heredity apply to human characters. This is being done by the Eugenics Record Office at Cold Springs Harbor, New York, under the patronage of the Carnegie Institution.

Specifically, the Record Office seeks pedigrees of families in which one or more of the following traits appear: short stature, tallness, cor-

pulency, special talents in music, art, literature, mechanics, invention, and mathematics, rheumatism, multiple sclerosis, hereditary ataxy, Ménière's disease, chorea of all forms, eye defects of all forms, otosclerosis, peculiarities of hair, skin, and nails (especially red hair), albinism, harelip and cleft palate, peculiarities of the teeth, cancer, Thomsen's disease, hemophilia, exophthalmic goiter, diabetes, alkaptonuria, gout, peculiarities of the hands and feet and of other parts of the skeleton.

In brief, then, the aim of eugenics is through heredity to give the individual the greatest of all birthrights, viz., good human protoplasm, and to eliminate, as far as may be possible, bad human protoplasm.

PRINCIPLES OF HEREDITY

For a clearer understanding of the hereditary transmission of disease, malformations, and defects it is necessary to have an understanding of the principal views upon organic evolution and the theories of heredity. The student of preventive medicine should especially have a clear comprehension of Mendel's work, which has thrown a flood of light upon the problems before us. Mendel has opened new vistas in biology, which have a practical bearing upon public health work. It is evidently impossible in a short space to do justice to such large subjects as evolution and heredity, and the student is, therefore, referred to the authorities given at the end of this chapter, which will repay careful study.

Variation.—It has been a matter of common observation that like *tends* to beget like rather than "like *begets* like," for there is a tendency toward new departures.

Two distinct sorts of divergences may appear among the members of a single family. The first is known as variation; the second as mutation.

By variation we understand those slight differences which invariably distinguish all the members of every family. They consist of individual variations which affect every part and every character. Such differences are also known as fluctuating, normal, or continuous variations to distinguish them from abnormal, definite, or discontinuous variations, which are more properly termed mutations. As examples of variation in man we may cite the variations in size or stature, color of skin and eyes, curliness of hair, configuration of face, etc.

Darwin lays particular emphasis upon the importance of variation in his views of organic evolution.

Darwin's Theory.—The Survival of the Fittest.—Darwin's views [1] of heredity are based upon his theory of organic evolution. Two

[1] Darwin: "The Origin of Species," "The Descent of Man," etc.

separate factors are primarily concerned: (1) the fact of fluctuating variation, that is, that no two members of the same family ever resemble one another exactly; and (2) the occurrence of a struggle for existence between organisms, owing to the geometric rate of increase of living things. From these two facts it follows that, when a change of environment takes place, certain members of an existing species will be somewhat better adapted than others to withstand the new conditions, and the former will tend to survive to the exclusion of the latter. Darwin assumes that during a long series of generations this process will cause a steady change in the character of the species in the direction of better adaptation to the new conditions. In other words, Darwin considers that an accumulation of a series of small changes due to the influence of environment are transmitted hereditarily through natural selection.

The remarkable effects produced in the case of domestic animals and plants by the action of artificial selection greatly influenced Darwin's views upon the selective influences which exist in nature. Darwin believed in the hereditary transmission of acquired characters and regarded organic evolution as proceeding by a slow, gradual, or continuous process. There can be no doubt but that natural and sexual selection have a great influence, but whether sufficient to originate new species or even new specific characters is a question. Now that the transmission of acquired characters is denied by students of heredity, and the fact that DeVries has actually observed new species arise suddenly, Darwin's theory of organic evolution and the origin of species is receiving critical examination.

Darwin firmly believed that the characters of organisms can be modified by selection, and he made this the foundation stone of his theory of evolution. The brilliancy of the mutation theory of DeVries, coupled with his great service to biology in rediscovering the Mendelian laws, has somewhat dazzled our eyes. Castle believes, after ten years of continuous work in selection, that much may be accomplished by this means quite apart from the process of mutation, and considers that the work of DeVries himself argues strongly in favor of this idea, although his interpretation of it is adverse to selection. From the evidence at hand we must conclude that Darwin was right in assigning great importance to selection in evolution, that progress results not merely from sorting out particular combinations by large and striking unit characters, but also from the selection of slight differences in the potentiality of gametes representing the same unit character combinations.

Mutation.—Mutations comprise definite differences, usually of considerable magnitude—differences that indicate specific characters or the beginning of new species. Such differences are also known as ab-

normal, definite, or discontinuous variations, but more properly they are termed mutations, sometimes "sports." Mutations may be either useful or harmful. They arise spontaneously and may be transmitted hereditarily in accordance with Mendel's law. As examples of mutations in man we may cite albinism, polydactylism, brachydactylism, etc.

DeVries, Bateson, and the "mutationists" are convinced that mutation is a much more important factor in the origin of species than variation, as understood by Darwin. In the light of Mendel's work mutations appear to be unit characters which arise "spontaneously"—in some instances they represent recessive characters that have remained dormant for many generations.

DeVries—Discontinuous Evolution.—The observations of DeVries upon the evening primrose (*Œnothera lamarckiana*) convinced him that species may arise suddenly, that evolution is discontinuous and goes by leaps and bounds rather than by the slow or continuous process of organic evolution described by Darwin.

Mutation is the term applied by DeVries to express the process of origination of a new species or a new specific character, when this takes place by the discontinuous method at a single step. DeVries believes that this is the most important, if not the sole, method by which new species or specific characters arise. To those who are convinced that acquired characters are not inherited the explanations of Lamarck and Darwin have always been incomplete. Darwin insisted that nature does not make jumps and that new species arise slowly through the action of natural selection on minute variations—a gradual or continuous evolution.[1] From his experiments DeVries concludes that when selection is really efficient the full possible effects of this process are exhausted in quite a small number of generations, and that then the only further effect of selection is to keep up the standard already arrived at. DeVries actually obtained quite a number of new types of plants which arose suddenly and naturally. When they made their appearance the majority of the new types came true to seed. With regard to the causes of mutation little is known, unless we assume that they represent unit characters which have long remained recessive.

Weismann's Views.—Weismann's[2] views are based largely upon his assumption that the germ plasm is distinct from the body and that acquired characters are not inherited. The parent is composed biologically of somatic or body cells which are mortal, and reproduction cells or germ plasm which is distinct, continuous, immortal. The germ cells undergo the least modification from their original condition. Indeed, Weismann believes that there is no reason for supposing that they

[1] Darwin, however, recognized the facts of mutations or "sports" as he called them and dwelt upon their importance.

[2] Weismann, A.: "Essays upon Heredity," 1889, and "The Evolution Theory," 1906.

have undergone any modification at all. From this point of view we may consider the nature of a given series of animals as being determined only by the particular series of cells which constitute the direct ancestry of the germ cells in each individual. The cells which make up the bodily structure may be regarded as the result of so many offshoots which come to an end at the death of the organism and have no progeny of their own.

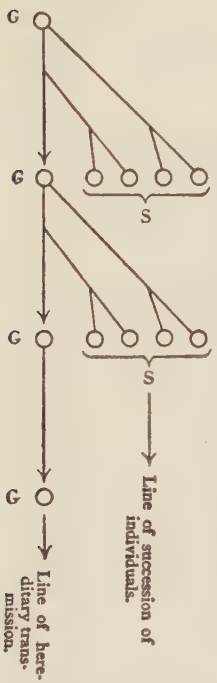


FIG. 59.—WILSON'S THEORY OF INHERITANCE MODIFIED BY LOCK (G, germ cells; S, somatic cells).

The minute study of the germ cells taken in connection with modern experimental work on the methods by which inheritance takes place shows a strong tendency to confirm Weismann's view, so far as the inheritance of distinct and definite characters is concerned.

Wilson[1] has expressed Weismann's theory as follows: It is a reversal of the true point of view to regard inheritance as taking place from the body of the parent to that of the child. The child inherits from the parent germ cell, not from the parent body, and the germ cell owes its characters not to the body which bears it, but to its descent from a preëxisting germ cell of the same kind. Thus, the body is, as it were, an offshoot from the germ cell. As far as inheritance is concerned, the body is merely the carrier of the germ cells which are held in trust for coming generations. Fig. 59 illustrates Wilson's theory of inheritance as modified by Lock.

Mendel's Law.—We are indebted to Mendel[2] for one of the most important observations of biology—the most important, in fact, with reference to heredity. The essential factors of Mendel's discovery are: (1) unit characters, (2) dominance, (3) segregation. By a unit character is understood any characteristic of an individual that is transmitted from parent to offspring through

[1] Wilson: "The Cell in Development and Inheritance," p. 13.

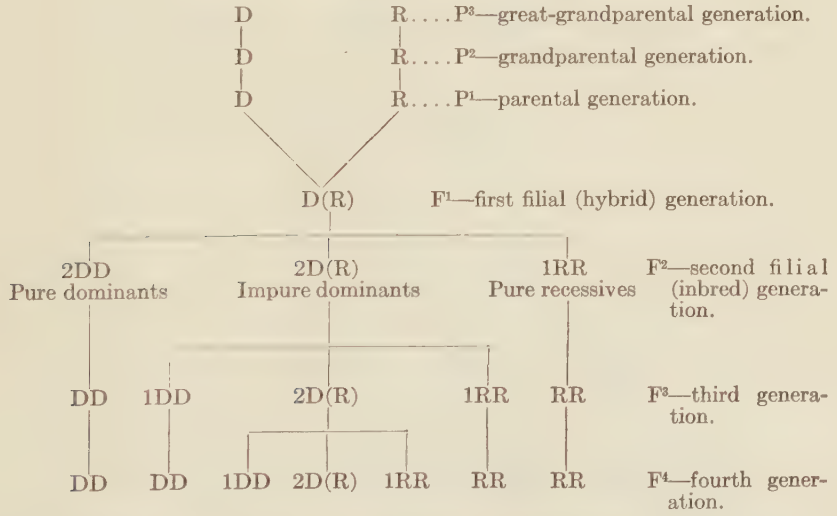
[2] Gregor Johann Mendel was born July 22, 1822, at Heizendorf in Austrian Silesia. In 1843 he entered the Augustine Convent at Altbrunn as a novice, and was ordained priest in 1847. Mendel was a teacher of natural science in the Brunn Realschule from 1853 to 1868, when he was appointed abbot of his monastery.

Mendel published only the results of his work upon hybridization with peas and a few of his experiments with *Hieracium*. The original paper on "Hybridization" was published in the *Verh. Naturf. Ver.* in Brunn, Abthandlungen IV, 1865, which appeared in 1866; the paper on "Hieracium" appeared in the same journal, VIII, 1869. The student is advised to read "Mendel's Principles of Heredity" by W. Bateson, 1909, in which he will find a translation of these two important papers. A clear exposition is also given by R. C. Punnett in his book entitled "Mendelism" (1911).

successive generations and which conforms to the following: they are usually complementary. When parents with complementary unit characters unite, it is found that one character predominates over the other. This is known as dominance. It has further been found that the unit characters contributed by the respective parents do not, as a rule, blend, but remain separate or distinct. This is known as segregation. The principles of segregation and dominance have been found to apply to the inheritance of many characters in animals and plants. It should be carefully borne in mind that the unit characters themselves are not transmitted as such in the germ cells. Just what is transmitted is not definitely known. It is quite sure that the only thing that is inherited in the germ cells is something which determines the development of the unit character. This something is called a determiner.

The essence of this great discovery was published by Mendel in a short paper in 1866. By some extraordinary chance Mendel's observations were entirely lost sight of until the same facts were independently rediscovered in 1899 by DeVries, working in Holland, by Correns in Germany, and by Tschermak in Austria.

A Schematic Representation of Mendel's Law



D and R represent complementary unit characters, D the dominant character, and R the recessive character. D(R) represents a dominant with the recessive character unexpressed but potentially present. DD means pure dominants, and RR pure recessives.

Mendel's law may best be understood from a concrete illustration. One of the simplest cases is that of the heredity of color in the Andalusian fowl, which has been so clearly described by Bateson.

There are two established color varieties of this fowl: one with a great deal of black and one that is white with some black markings or splashes. For convenience we may refer to these as the black and white varieties respectively. Each of these breeds true by itself. Black mated with black produce none but black offspring. White mated with white produce none but white offspring. Crossing black and white, however, results in the production of fowls with a sort of grayish color called "blue" by the fancier, though in reality it is a fine mixture of black and white. If we continue to breed succeeding generations from these blue hybrid fowls we get three different colored forms. Some will be blue, like the parents, some black, like one grandparent, some white, like the other grandparent. Further, these different colors appear in certain definite proportions among the three classes of descendants. Of the total number of the immediate offspring of the hybrid blues, approximately one-half will be blue, like the parents, approximately one-fourth black, and one-fourth white, like each of the grandparents. Thus, black bred together produce only blacks; the white similarly produce only whites; the blues, on the other hand, when bred together produce a progeny sorting into three classes, and in the same proportion as that produced by the blues of the original hybrid generation. The fact that the black grandchildren and the white grandchildren respectively breed true is a very important fact. In this illustration no race of the hybrid blue character can be established, for the blues always produce blacks and whites as well as blues (see diagram).

FIG. 60.—DIAGRAM SHOWING THE COURSE OF COLOR HEREDITY IN THE ANDALUSIAN FOWL, IN WHICH ONE COLOR DOES NOT COMPLETELY DOMINATE ANOTHER. *P*, parental generation. The offspring of this cross constitute F_1, the first filial or hybrid generation. F_2, the second filial generation. Bottom row, third filial generation. (Kellicott.)

Another instance which illustrates the phenomenon of dominant and recessive characters as well as segregation is here given. If black and white varieties of guinea pigs are crossed the offspring are all black,

like one parent; that is, when black and white characters are brought together in the guinea pig, these do not appear to blend into gray or "blue," as in the case of the Andalusian fowl, but one character alone appears. The black seems to cover up or wipe out the white. The black color is, therefore, said to be dominant and the white recessive. The white character, however, has not disappeared, for when the black offspring are crossed together the progeny falls into two groups: some black and some white. Three-fourths of the progeny are black; that is, they resemble the hybrid form and at the same time one of the grandparents, while the remaining fourth resemble the other white grandparent. Some of these blacks will breed true and are, therefore, known as homozygotes. Some of the blacks contain a mixture of the black and white characters and are, therefore, known as heterozygotes. The hereditary transmission of the color character in these two illustrations through the germ cell is shown in the accompanying diagram.

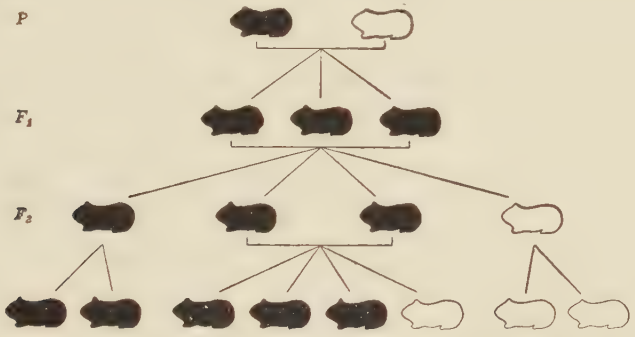


FIG. 61.—DIAGRAM SHOWING THE COURSE OF COLOR HEREDITY IN THE GUINEA-PIG, IN WHICH ONE COLOR (BLACK) COMPLETELY DOMINATES ANOTHER (WHITE). Reference letters as in Fig. 60. (Kellicott.)

Unit characters may either be positive or negative; that is, they may be due to the presence or absence of "something" in the germ cell or sperm cell. This something, known as a determiner, is a force, a molecular structure or an enzyme (?) in the nuclear matter of the germ-plasm. Thus, the determiner in the case of pigment is not the pigment itself, but something that activates pigment production. These determiners are transmitted in the germ plasm and are the only things that are truly transmitted. The determiner may be either in the ovum or the sperm.

An hereditable character may be due to the presence or absence of a determiner in the germ plasm of both parents. When a character is due to the presence of a determiner it is called positive, when due to the absence of a determiner, negative. Thus, a brown eye depends on a determiner that produces the brown-colored pigment, while the blue eye depends upon the absence of such a determiner. It is not always

easy to anticipate whether a given character is positive or negative. For instance, long hair in Angora cats, sheep, or guinea pigs is apparently not due to a factor added to short hair, but rather to an absence of a determiner that stops growth in short-haired animals.

One of the most important conclusions from Mendel's observations is that the different inherited traits act independently; that is, they do not blend. In other words, the definitely hereditable characters act as independent units that are without any apparent relation to other peculiarities of the individual concerned. Furthermore, these units do not interfere with each other. It follows that all the unit characters of an individual are to be regarded as mutually independent assemblages. This is the doctrine of unit characters. According to this doctrine, each individual is of dual origin, paternal and maternal, and each individual is made up of a mosaic of inherited characters, some of which may be dominant, others recessive. The idea of unit characters capable of being inherited independently of one another is one of the most important conceptions which has been added to our knowledge of heredity. We now know from the phenomenon of segregation what constitutes purity in a strain of animals or plants; that is, purity does not depend upon the length of time during which a race has exhibited a constant character, for a strain of absolute purity may arise from the second generation of a cross. Mendel's law has not only explained many facts in heredity, but also has important practical bearing in the improvement of the breeds of cultivated plants and domestic animals.

Atavism and Reversion.—Atavism (from atavus, a grandfather) is the inheritance of properties not manifest in either parent, but present in the grandfather or some relatively recent ancestor. Mendel's observations upon recessive characters now make plain some of the phenomena known as atavism. According to Castle, atavism or reversion to an ancestral condition can be completely explained by the Mendelian principles. It is nothing more or less than the reassertion of recessive unit characters that have long been overshadowed by dominant characters. It seems that recessive characters may not be lost, no matter how long they remain latent or dormant.

The term "atavism" is sometimes employed to mean any reversionary condition, whether favorable or unfavorable, while the term "reversion" means a return in the offspring to a lower type, usually of some remote ancestor. The degenerations which run in families may be instances either of atavism or reversion, or mutation.

Darwin's classical experiment illustrating reversion consisted in crossing a barbed fan-tail female pigeon with a barbed spot male and producing offspring hardly distinguishable from the wild Shetland species of blue-rock pigeon (*Colomba livia*). This is a case of reversion,

in which an artificially bred and highly specialized race quickly recovered characters which had been lost during many generations. A foal is sometimes born with a few stripes on its forelegs, as if reminding us of striped wild horses. Highly cultivated and specialized flowers and vegetables have a tendency to revert, and sometimes produce forms hardly distinguishable from their wild progenitors. Reversion is due to the reassertion of latent ancestral characters. It is an impelling hereditary force which must be taken into account. True reversion may arise in pure bred races, but is much more frequent as the result of hybridization.

The facts of reversion and atavism are of peculiar interest to man, for the reason that the human species has, through unconscious selection and conscious effort, improved the race to its present point of superiority. Whether civilized man to-day is superior to ancient races may be doubted, but the fact is plain that civilization is breeding an artificial and highly civilized strain that shows artificial departures from primitive stock.

It is well known that the high bred and "fancy" races of the domesticated animals show a marked tendency to reversion or deterioration of type. Likewise, the human race shows the same tendency to revert to types resembling its forebears. The present level attained by the more highly civilized races can only be maintained by a continuation of that struggle for improvement, progress, and desire for perfection which is an inborn characteristic and an essential element of progress. Owing to the artificial position to which the human race has brought itself, it becomes necessary to continue the struggle—to stand still means rapid deterioration. Some of the stigmata of degeneration and hereditary defects may be accounted for by this natural tendency on the part of an artificially nurtured standard to slip backward.

Galton's Law of Filial Regression.—Filial regression has nothing to do with reversion. The law of filial regression concretely stated is that offspring are not likely to differ from mediocrity in a given direction so widely as their parents do in the same direction. There is a continual tendency to sustain a specific average or a stock average.

Let us take a simple instance from Professor Karl Pearson's "Grammar of Science." Suppose a group of fathers with a stature of 72 in.: the mean height of their sons is 70.8 in.—a regression toward the mean height of the general population. On the other hand, fathers with a mean height of 66 in. give a group of sons of mean height 68.3 in.—again nearer the mean height of the general population. The "regression" works both ways—there is a leveling up as well as a leveling down. "The father with a great excess of the character contributes sons with an excess, but a less excess of it; the father with a great

defect of the character contributes sons with a defect, but less of it" (Thompson).

THE CELL IN HEREDITY

Each parent (male and female) is composed biologically of somatic or body cells, which are mortal, and germ plasm which is distinct, continuous, immortal. The development and embryology of the germ and sperm cells are of particular interest to the student of heredity.

The view has gained ground and general acceptance that the nucleus is the chief or exclusive bearer of the hereditable characters; that is, the female nuclear material transmits the characters of the mother and her forebears and the male nucleus those of the father and his forebears to the offspring.

Cells divide and multiply in two ways: (1) by direct division or amitosis, and (2) by indirect division or mitosis. Direct division occurs more frequently than is usually suspected. The process appears to be a very simple one; the nucleus divides without any preliminary arrangement of its structure, the cytoplasm is constricted, and presently we have two cells in place of one. Indirect division or mitosis appears to be the natural mode of cell development. The chromatin, which is the deeply staining matter in the nucleus, rearranges itself from its "resting" stage. After a complicated process the nuclear matter forms itself into a long cylindrical thread known as the linene thread. This then divides into links or chromosomes.[1] The chromosomes are of special interest, for they are believed to carry the hereditable traits.

In amitotic division each chromosome is divided in half longitudinally, as a stick might be split up the middle, and after a very complex process the halves of each split chromosome migrate to opposite poles. Then each centrosome attracts a group of chromosomes consisting of just one-half of the original chromatin material. Each group then, in orderly fashion, rounds itself into a new nucleus, and the body of the cell (the cytoplasm) constricts across the equatorial plane, and two cells are formed.

Every species of plant or animal has a fixed and characteristic number of chromosomes which regularly recurs in the division of all of its cells and in all forms arising by sexual reproduction the number is even. Thus, in some of the sharks the number of chromosomes is 36, in certain gastrapodes it is 32; in the mouse and salamander, the trout, the lily, 24; in the worm *Saggita*, 18; in the ox and guinea pig, 16; in man the number was formerly stated as 16, now 24. In

[1] For a full understanding of cell division the student is referred to one of the standard text-books upon Cytology, or Minot's "Embryology"; also, to E. B. Wilson's "The Cell in Development and Inheritance," 2d Ed., 1900.

crustaceans the number of chromosomes may be as high as 168. In a few insects the females have in their body cells one chromosome in addition to the number possessed by the males. This has been interpreted as bearing upon the determination of sex.

Van Beneden in 1885 discovered the important fact that the nucleus of the ovum and the nucleus of the spermatozoon which unite in fertilization contain each one-half of the number of chromosomes characteristic of the body cells.

As both the germ and sperm cells contain only half the number of chromosomes, a reduction must take place in the history of these cells; in fact, alike in the history of the germ cell and in the history of the sperm cell, there is a parallel reduction in the number of chromosomes to one-half. This reduction appears to be a preparation of the reproduction cells for their subsequent union, and a means by which the number of chromosomes is held constant in the species.

In sexual reproduction each centrosome attracts a group of chromosomes, half of which are of paternal origin and half of maternal origin. This is interpreted as meaning that the paternal and maternal chromosomes that unite to form the new zygote probably carry the hereditable characters.

The gist and meaning of the whole process to the student of heredity is the precisely equal partition of the maternal and paternal contributions, so that each of the daughter cells has a nucleus half from the mother and half from the father.

Although the ovum is much larger than the spermatozoon, each contributes equally so far as the amount of nuclear matter is concerned; the new individual is dual in its origin, and the offspring is a double creature and retains its duality to its dying day, and transmits it to succeeding generations.

Professor E. B. Wilson states the generally accepted opinion somewhat as follows: As the ovum is much larger it is believed to furnish the initial capital—including, it may be, a legacy of food yolk—for the early development of the embryo. From both parents alike comes the inherited organization which has its seat (according to most biologists) in the readily stainable chromatin rods of the nuclei. From the father comes a little body, the centrosome, which organizes the machinery of division by which the egg splits up and distributes the dual inheritance equally between the daughter cells.

The ovum may be stimulated to maturation without the sperm cell (parthenogenesis). When this happens individuals are produced similar to, but not as vigorous as, the normal types. The sperm cell similarly is able to develop without the nuclear matter of the egg. In other words, the ovum and the sperm each contain potential factors for the new individual. As we have already seen, in accordance with

Weismann's theory, that the germ plasm is independent of the body and is continuous; therefore, acquired characters not affecting the germ plasm are not inherited in accordance with this conception.

Foreign bodies carried along by either the germ or sperm cells are not instances of true heredity; therefore, in the present-day conception of heredity it is not possible for a microbic disease to be transmitted hereditarily, even though the microorganism is contained in either the germ or the sperm. Thus, hens may be caused to lay colored eggs by feeding the hen with anilin dyes. Anaphylaxis is an example of a transmitted property, but the substance, whatever it is, seems to be carried along with the maternal germ cell as a foreign body. In the case of syphilis, the *Treponema pallidum* may be carried along by the germ or sperm, and the disease is said to be transmitted hereditarily, but, strictly, the microorganism is carried as a foreign body and not as a unit character or constituent part of the nuclear matter.

BIOMETRY

Statistical methods applied to biology have been termed biometry by Professor Karl Pearson. Francis Galton's book on "Natural Inheritance" is a pioneer in the subject, and embodies a lucid introduction to the statistical study of variation and inheritance. The health officer must be familiar with statistical methods not only in their application to biology, but as they relate to vital statistics. The health officer who lacks the quantitative view or who fails to grasp the statistical values of the facts and factors in preventive medicine works under a decided handicap. The sanitarian who is ignorant of statistical methods must necessarily grope in the dark. Efficiency and economy in public health work depend not alone upon a knowledge of the biological sciences, but also upon a correct sense of proportion. The statistical method is a strong lever which makes for sane administration, economy in expenditure, efficiency of effort; in short, successful results.

Statistics deal with groups rather than with individuals. It must be understood that the average of a group may represent something quite different from any individual which the group contains. Also a group may contain individuals of very diverse natures. In collecting statistical material the data must be gathered without any preconceived ideas and without neglecting any members. In this respect statistical methods differ from biological methods, which require careful discrimination of data.

The quantitative determination of a character may be made by various methods, as by counting or by measurement.

The statistical method may be illustrated by a simple model, such as that suggested by Galton. This is a modification of the familiar

bagatelle board covered with glass and arranged as shown in Fig. 62. A funnel-shaped container at the top of the board is filled with peas or similar objects. Below this is a regular series of obstacles symmetrically arranged, and at the bottom of the board is a row of vertical compartments also arranged symmetrically with reference to the chief axis of the whole system. If we allow the peas to run through the funnel and fall among the obstacles into the compartments below, we find that their distribution will follow certain laws capable of precise mathematical description. The distribution of the peas may be predicted

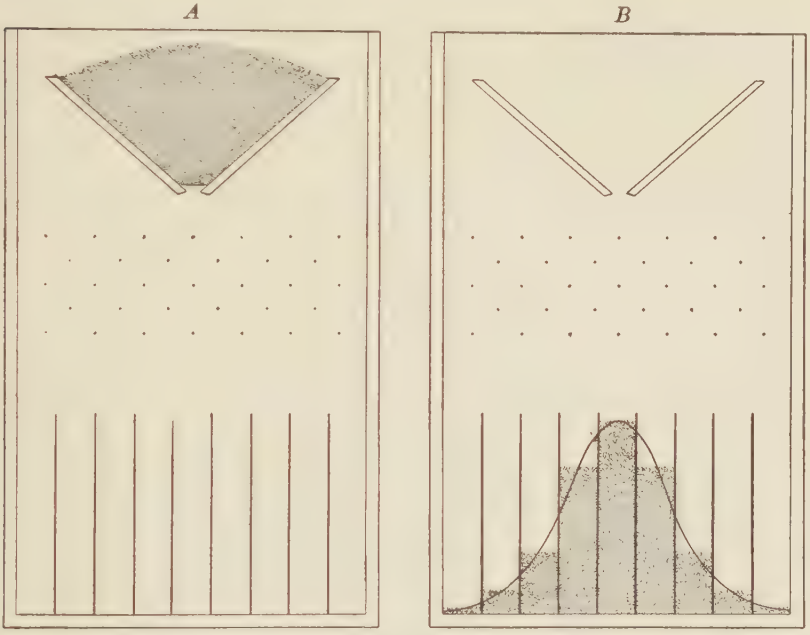


FIG. 62.—MODEL TO ILLUSTRATE THE LAW OF PROBABILITY OR "CHANCE." *A*, Peas held in container at top of board. *B*, Peas after having fallen through the obstructions into the vertical compartments below. The curve connecting the tops of the columns of peas is the normal probability curve.

with fair accuracy. The middle compartment will receive the most; the compartments next the middle somewhat fewer; those further from the middle still fewer; and the end compartment fewest. If we connect the top of each column of peas by a curved line we get a curve known as the "normal frequency curve." A curve of the same essential character would result from plotting the dimensions of a thousand cobblestones, the deviation from the bull's eye in a target shooting contest, or by plotting the variability of a biologic character, such as the stature or strength of men, the spread of sparrows' wings, the number of rays on scallop shells, or of ray flowers of daisies.

While from the above law of probability we know quite definitely

what the general distribution of the peas will be, we do not know at all the future position of any single pea. Of this we can speak only in terms of probability. The chances are very high that it will fall in one of the three middle compartments, very low that it will be one of the extreme compartments. The chances are equal that any individual pea will fall above or below the average or middle position. We therefore see that in any group there are many more individuals near the average than there are in the classes removed from the average, and the farther the removal of a class from the average the smaller the number of individuals in that class; hence, we have the important fact in statistical methods that an individual may belong to a group without representing it fairly. In order to get a correct idea of the whole group we must know first to what extent deviation in each direction

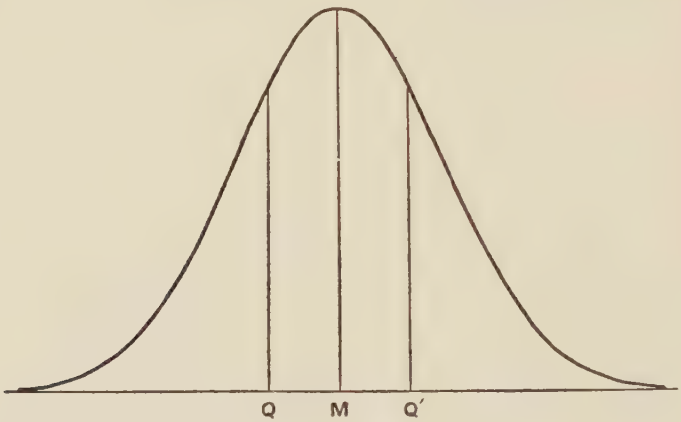


Fig. 63.—Normal Curve. (Lock.)

occurs above and below the group average; and, second, the average amount by which each individual of the group deviates from this group average; that is, we must know the amount of variability as well as the extent of the greatest divergence from the average. Hence, we have the following definitions and corollaries:

The *mode* of a normal curve is the longest perpendicular which can be drawn from the base line to meet the curve itself, M, Fig. 63. The normal curve is symmetrically on either side of the mode; that is to say, two perpendiculars drawn from the base to the curve on either side of the mode and at the same distance from it will be equal in length.

The *median* is a perpendicular line which divides the area of the curve into two equal halves. In dealing with a symmetrical curve the position of the mode is identical with that of the median.

The *mean* or average of all the values from which the curve is constructed is the foot of the median. In any actual case obtained by

practical methods the position of the mode, the median, and the mean will only be approximately the same because such a curve is never perfectly symmetrical.

The *quartile* is the distance from the median to a perpendicular line extending from the base of the curve at such a distance from the median that it divides the area inclosed by the median, the base, and half the curve into two equal parts. Any given curve will have two quartiles, one on either side of the median. They are shown at Q and Q′. (Fig. 63.)

A *variate* is one of the separate numerical values from which a curve of variability can be constructed. The accuracy of the statistical method is usually proportionate to the number of variates out of which the curve is built. The biometrician usually deals with some such number as 1,000 variates. The total number of variates is represented by the area inclosed by the curve, and it will be seen that half the total number of variates falls between the two quartiles and half outside of them.

A *class* may be defined as a group of variates all of which show a particular value or a value falling between certain limits.

The *frequency of a class* is the number of variates which it contains.

The *amount of variation* shown by a particular group of variates is measured by the degree of slope of the curve. A flat curve indicates greater variability and a steep curve denotes less variability.

The *standard deviation* of a normal curve is the measure of variability and is more often used than the quartile and is expressed shortly as σ. The value of σ is found by multiplying the square of the deviation of each class from the mean (or mode) by the frequency of the class, adding together the series of products so obtained, dividing this number by the total number of variates, extracting the square root of the result, and multiplying by the number of units in the class arranged.

The *coefficient of variability* is a purely abstract number obtained by dividing the standard deviation by the magnitude of the mean in any particular case and multiplying the result by 100.

The *probable error* arises from the circumstances that half the total number of variates lies outside the limits of the quartile and half within. The probable error of any statistical determination is obtained by finding a pair of values lying one above and one below the true value required. For further details regarding properties of normal curves the student is directed to Davenport's "Statistical Methods with Special Reference to Biological Variation."

HEREDITY VERSUS ENVIRONMENT

How much of our physical and mental makeup is due to heredity (nature) and how much to environment (nurture) is one of the much-discussed problems. It seems evident to students of biology that by far the overwhelming factor in our organization is set and definitely fixed at our birth. Heredity appears to be the overshadowing influence of first and prime importance. Herbert Spencer well said that "inherited constitution must ever be the chief factor in determining character." Environment may influence the individual, but apparently has small and slow power of propagating itself for good; great and rapid power for evil. That is, the hereditary transmission of acquired characters is denied, but the transmission of defects of organization, such as insanity, deaf mutism, the consequences of syphilis, alcoholism, and other vices, are fully recognized. Atavism, reversion, and mutations must not be regarded as instances of the hereditary transmission of acquired characters in the biological sense. The tendency of the artificially bred strains of the civilized human races to revert and deteriorate has already been emphasized.

Despite the teachings of biology we are convinced that life is inexorably conditioned by its environment. Jordan states that "among the factors everywhere and inevitably connected with the course of descent of any species variation, heredity, selection, and isolation must appear; the first two innate, part of the definition of organic life; the last two extrinsic, arising from the necessities of environment, and not one of these can find leverage without the presence of the others." In the present state of our knowledge, while we are convinced that heredity plays the major rôle, we are by no means prepared to deny the influence of environment.

IMMUNITY GAINED THROUGH INHERITANCE

Immunity to disease is either natural or acquired. Natural immunity is inherited through successive generations of a species or a race. Acquired immunity, like other acquired characters, is probably not inherited as a "unit character" in the sense of Mendel. Thus, there has been little variation in our natural power to resist most infections, such as tuberculosis, yellow fever, plague, smallpox, cholera, tetanus, measles, scarlet fever, diphtheria, and so on through a long list, although these diseases have doubtless afflicted the human species through untold ages. The fluctuating virulence of some infections is a matter of common knowledge, and is doubtless due to many factors. In a few well-known instances a certain amount of tolerance or re-

sistance has been gained and perhaps transmitted through succeeding generations by a process of the survival of the fittest. Thus, syphilis is much less virulent now than it was during the great pandemic of the sixteenth century. The resistance which the natives enjoy to malaria in badly infected quarters of the globe is largely acquired as a result of early infections, and this increased resistance is perhaps partly transmitted by a weeding out of the very susceptible (see chapter on Immunity).

CHAPTER III

THE HEREDITARY TRANSMISSION OF DISEASE

We are now prepared to discuss more in detail the hereditary transmission of disease. The question whether disease is ever transmitted hereditarily or not rests somewhat upon our conception of disease; that is, whether it is an entity, a process, or a "unit character." The process itself, of course, cannot be transmitted, but the potentiality of it may be involved in some peculiarity in the organization of the germ plasm. This may be, and often is, transmitted through successive generations. In the limited sense in which the word "heredity" is used in biology and in the limited sense in which the word "disease" is used in pathology, there may be no inherited diseases, but this appears to be a quibble of words or a matter of definitions. While we are not familiar with the intimate processes concerned, we are certain that many abnormal conditions of mind and body are transmitted. Some of them follow the Mendelian principles.

Formerly a large number of diseases were regarded as transmissible, but the list has been revised and restricted as a result of recent studies. The reappearance of a diseased condition in successive generations does not prove that it has been transmitted or even that it is transmissible. This mistake has been made with tuberculosis and other infections.

Lack of completeness vitiates most of the statistics bearing on heredity in relation to human diseases. Even in the case of clearly inherited diseases there are very few pedigrees sufficiently complete for the study of the applicability of Mendelian and other laws of heredity.

Sometimes the disease itself is not transmitted, but a tendency to the disease is transmitted. This will be discussed again.

Some unit characters as well as certain diseases are transmitted hereditarily, but limited to one sex; that is, the disease or condition appears in one sex only, although transmitted by the other. The best example of a sex-limited disease is hemophilia, which affects males almost exclusively, but is transmitted through the normal female. Color-blindness is also transmitted hereditarily, but is sex-limited, as it affects males almost exclusively.

This remarkable sort of inheritance, known as sex-limited inheritance,

occurs when the male parent is characterized by the absence of some character of which the determiner is typically lodged in the sex (x) chromosome. A striking feature of this sort of heredity is that the trait appears only in males of the family, but is not transmitted by them; it is transmitted, however, through normal females of the family. Examples of this sort of heredity occur in hemophilia, color-blindness, also in multiple sclerosis, atrophy of the optic nerve, myopia, ichthyosis, and muscular atrophy. The explanation is the same in all cases of sex-limited heredity. The abnormal condition is due to the absence of a determiner from the male sex chromosome.

The diseases, defects, and conditions believed to be transmitted hereditarily are discussed in the following pages. Some of these diseases, malformations, and defects of organization follow Mendel's law. It is probable that other diseases, tendencies, and characters are transmissible, but the subject has only recently been placed upon a scientific basis, and it will require careful and prolonged observation to establish the facts. It is often difficult to determine whether the disease itself or a tendency to the disease has been transmitted in any particular case, and, further, it is often difficult to decide whether an individual has inherited or acquired his affliction.

The transmissible defects which are of principal concern to the human species are the defects of organization of the central nervous system. It is important to remember that the defects of the nervous system do not necessarily propagate just the same defects in the succeeding generations. Thus, an epileptic does not necessarily beget epileptics; epilepsy, insanity, degeneracy, color-blindness, and other stigmata may arise as the result of deficiencies of various kinds in the forebears.

Defects such as harelip, cleft palate, cervical fistula, spina bifida, etc., are not true instances of hereditary transmission of specific characters. They rather represent an inherited deficiency in developmental vigor. These defects for the most part represent the failure of parts to unite during embryological development; in other words, the failure of embryological clefts to close normally. Such deformities, as well as clubfoot, web fingers, and other acquired or congenital deformities or disfigurations, are not, as a rule, transmitted.

Some practical problems of great importance arise from our knowledge of the hereditary transmission of disease and defects. A man or woman who intends marrying is now more than justified in carefully examining the personal and medical histories of the family of his or her intended mate. It is not only possible to foretell the color of the eyes, the nature of the hair, and other Mendelian characters in the future offspring, but it is also possible to foretell, with mathematical precision, the chances of transmitting defects, such as insanity, epi-

lepsy, degeneracy, deaf-mutism, color-blindness, migraine, and other nervous disorders, as well as hemophilia, polydactylism, brachydactylism, albinism, and other stigmata. In any doubtful case it may be well to consult a student of heredity, for it is possible to foretell with precision in certain cases which characters will and which will not be transmitted.

To illustrate the precision with which the characters of offspring may be predicted in the best studied cases, we need only refer to the color of the eyes. Two parents with pure blue eyes will have only blue-eyed offspring, for they both lack the brown pigment which determines the color of the iris. Similarly, if the hair of parents be flaxen, this may be taken as evidence of the absence of a hair-pigment-determiner in the germ plasm, and the offspring will have flaxen hair. For the same reason parents with lack of curliness or waviness of hair will have only straight-haired children.

In determining whether transmissible characters are apt to reappear in successive generations or not we must know whether these characters are positive or negative, that is, whether they are due to the presence or absence of determiners.[1]

Inbreeding may be hazardous, for reasons that are well understood. The marriage of cousins will be evidently hazardous if the objectionable hereditary characters are dominant, for in this case the danger is plain; if the characters are recessive the danger is specially unfortunate, because of unexpected outcroppings in the offspring. Inbreeding tends to secure homozygous combinations, and this brings to the surface latent or hidden recessive characters. Crossbreeding brings together differentiated gametes which, reacting on each other, produce offspring of greater vigor. On the other hand, continued crossbreeding only tends to hide inherent defects, not to exterminate them; inbreeding only tends to bring them to the surface, not to create them. It is not, therefore, correct to ascribe to inbreeding by intermarriage the creation of bad racial traits, but only their manifestation. Further, a racial stock which maintains a high standard of excellence under inbreeding is certainly one of great vigor and free from inherent defects (Castle).

The variety of the product of consanguineous marriage is well brought out when we compare localities. Thus, consanguinity on Martha's Vineyard results in 11 per cent. deaf mutes and a number of hermaphrodites; in Point Judith, 13 per cent. idiocy and 7 per cent. insanity; in an island off the Maine coast the consequence is "intellectual dullness"; in Block Island, loss of fecundity; in some of the "Banks" off the coast of North Carolina suspiciousness and an inability

[1] We do not yet know all the unit characters in man, and it is impossible to foretell which of them are due to positive determiners and which to the absence of such.

to pass beyond the third or fourth grade of school; in a peninsula on the east coast of Chesapeake Bay the defect is dwarfness of stature; in George Island and Abaco (Bahama Islands) it is idiocy and blindness (G. A. Penrose, 1905). There is thus no one trait that results from the marriage of kin; the result is determined by the specific defect in the germ plasm of the common ancestor.

The Microbic Diseases.—It seems a confusion of thought to the student of heredity to speak of the inheritance of any microbic disease. At one time the hereditary transmission of microbic diseases was generally believed. Now we know that, in the true sense of the term, no infectious disease is transmitted hereditarily—for even in the case of syphilis the *Treponema pallidum* is carried in the germ or sperm as a foreign body. Tuberculosis at one time was considered as transmitted, but we now know that this occurs so seldom that the popular pamphlets are entirely justified in denying it entirely. Children are sometimes born with smallpox, measles, and other infections; these are not true instances of heredity, but cases of congenital transmission.

Congenital Transmission.—Prenatal infection is not a true instance of inheritance. Microbic diseases may be acquired by infection through the placenta during the fetal period. The placenta is a better filter for some infections than for others. Thus, anthrax and tuberculosis of the mother are rarely transmitted to the fetus, while there is great liability in the case of syphilis. The fetus *in utero* may take smallpox, measles, and other infections, but these instances are more properly spoken of as congenital than inherited.

We must remember that to be inherited on the part of the offspring or transmitted on the part of the parents biology includes only those characters or their physical bases which were contained in the germ plasm of the parental sex cells (Martius); or, as Verco says, "what operates on the germ after the fusion of the sex nuclei, modifying the embryo, or even inducing an actual deviation in the development, cannot be spoken of as inherited. It belongs to the category of early acquired deviations which are, therefore, frequently congenital."

Hereditary Transmission of a Tendency to a Disease.—While the disease itself may not be transmitted, a tendency to a disease, known as a diathesis, may be transmitted through successive generations. A person may inherit a small bony structure, a poor musculature, "weak" lungs, susceptible mucous membranes, an abnormal amount, distribution, or development of lymphoid structures, etc. In fact, we are not all born equal, and most persons have some vulnerable structure or organ which is commonly spoken of as their "weak point." In many cases this *locus minoris resistentiæ* is inherited as a defect in structure or function.

Davenport has collected the health records and other characteris-

tics furnished for over two hundred families by members of the families concerned. He finds certain definite facts in the behavior of some of the commoner diseases. As an example of the inheritance of a general weakness in an organ he cites the case of the mucous membranes. Thus, in one family the principal diseases to which there was liability were located in the mucous membranes of the nose, throat, and bronchi. In another family the center of susceptibility was more specific, being nearly confined to the nose and throat. In another family the weakness was in the ear; in another the lungs; in another the skin; in one family the kidneys were the seat of incidence, etc.

The examination of the health pedigrees of a number of families impresses one by the fact that the incidence of disease is not always haphazard, for in any large family the various causes of death do not occur in the proportions given in the census table for the population as a whole.

Tuberculosis.—We know that tuberculosis is never transmitted hereditarily, and is seldom contracted congenitally. The reason that tuberculosis runs in a family is twofold: (1) an inherited predisposition to the disease, and (2) increased chances of infection. Just what the tendency or predisposition is is not well understood. We do know, however, that the predisposition is not so great but that it may be overcome; the infection may be avoided and the disease prevented.

It is now perfectly plain that the principal reason why tuberculosis runs in families is the close association between the infected and well members of the family, which increases the chances of infection and reinfection.

All persons inherit more or less powers of resisting tuberculosis. The inborn immunity is not marked in any case; in some individuals it is quite feeble. The border line between immunity and susceptibility to tuberculosis in the human species is delicately balanced and may readily be overturned (see page 135).

Syphilis.—Syphilis and the consequences of syphilis are transmitted from parent to offspring—"even unto the third and fourth generation." Strictly speaking, and in accordance with the present-day conception of heredity, it may not be proper to speak of syphilis as a true instance of heredity, but whatever the definition of words may be the facts are plain. The reason that the student of biology refuses to regard syphilis as well as other microbic diseases as true instances of heredity is that the *Treponema pallidum* is transmitted in the germ plasm as a foreign body, and not as a unit character. The transmission of syphilis, therefore, does not obey Mendel's law. It must be remembered that while syphilis itself is not a true instance of hereditary transmission, the consequences of syphilis may descend as inherited defects through many generations. Syphilis may be transmitted in three ways:

(a) from the father (sperm inheritance); (b) from the mother (germ inheritance); (c) placental transmission (congenital). Osler summarizes the hereditary transmission of syphilis as follows:

(a) *Paternal Transmission* (*Sperm Inheritance*).—This is the most common form—in which the father is infected, the mother being healthy. The *Treponema pallidum* has not yet been found in the sperm cell, but we do not know its life phases, and from what we do know of the life history of syphilis it seems probable that all the sperm cells are not infective. A syphilitic father may beget an apparently healthy child, even when the disease is fresh and full-blown. On the other hand, in very rare instances a man may have had syphilis when young, undergo treatment, and for years present no signs of disease, and yet his first born may show very characteristic lesions. The closer the begetting to the primary sore the greater the chance of infection. A man with tertiary lesions may beget healthy children. As a general rule, it may be said that with judicious treatment the transmissive power rarely exceeds three or four years.

(b) *Maternal Transmission* (*Germ Inheritance*).—While the father may not be affected, in a large number of instances both parents are diseased, the one having infected the other, in which case the chances of fetal infection are greatly increased. Heredity through the mother alone is much more fatal to the offspring than paternal heredity. It is a remarkable and interesting fact that a woman who has borne a syphilitic child is herself immune, and cannot be infected, though she may present no signs of the disease. This is known as Beaumès' or Colles' law, and was thus stated by the distinguished Dublin surgeon: "That a child born of a mother who is without obvious venereal symptoms, and which, without being exposed to any infection subsequent to its birth, shows this disease when a few weeks old, this child will infect the most healthy nurse, whether she suckle it, or merely handle and dress it; and yet this child is never known to infect its own mother, even though she suckle it while it has venereal ulcers of the lips and tongue." In a majority of these cases the mother has received a sort of protective inoculation, without having had actual manifestations of the disease. A child showing no taint, but born of a woman suffering with syphilis, may with impunity be suckled by its mother (Profeta's law).

(c) *Placental Transmission.*—The mother may be infected after conception, in which case the child may be, but is not necessarily, born syphilitic. If the infection is late in pregnancy, after the seventh month, the child usually escapes.

Osler and Churchman state that syphilitics may marry with safety after they have undergone three years of thorough treatment and have been without symptoms at least one year after treatment has ceased.

Cancer.—It will probably be a long time before the final word can be said concerning the influence of heredity in cancer. At present there is no proof that heredity plays a part in the causation of cancer, but trustworthy conclusions are not possible in the present incomplete state of our knowledge upon the subject.

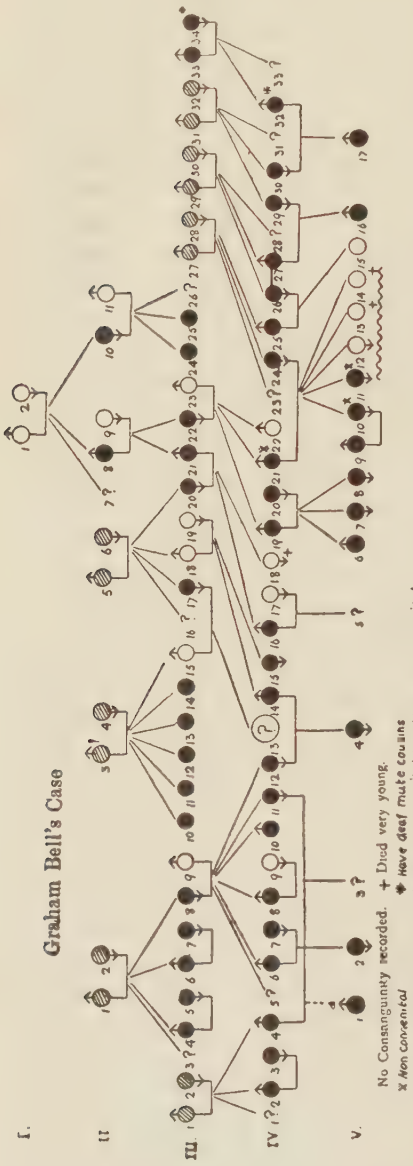


FIG. 64.—FAMILY HISTORY SHOWING DEAF-MUTISM. (From "Treasury of Human Inheritance.")

Leprosy.—Leprosy was formerly regarded as one of the inherited infections. Leprosy is not transmitted. The children of lepers born out of leper districts, in England or the United States, for example, never inherit it. The disease is contracted after birth, as tuberculosis and other microbic diseases are contracted.

Deaf-mutism.—Deaf-mutism is due to a great variety of causes, but in different individuals of the same family the chances are large that it is due to the same defect. This defect is frequently recessive, that is, hidden in the normal children. Two such normal children who are cousins but from deaf-mute stock tend to have about one-fourth of their offspring deaf-mutes. The proportion of congenital deaf offspring is thrice as great among cousin marriages as among others. The conclusions of Fay, based on extensive statistics, deserve to be widely known. "Under all circumstances it is exceedingly dangerous for a deaf person to marry a blood relative, no matter whether the relative is deaf or hearing, nor whether the deafness of either or both or neither of the partners is congenital, nor whether either or both or neither have other deaf relatives besides the other partner."

Albinism.—Albinism belongs to a class of cases resulting from the

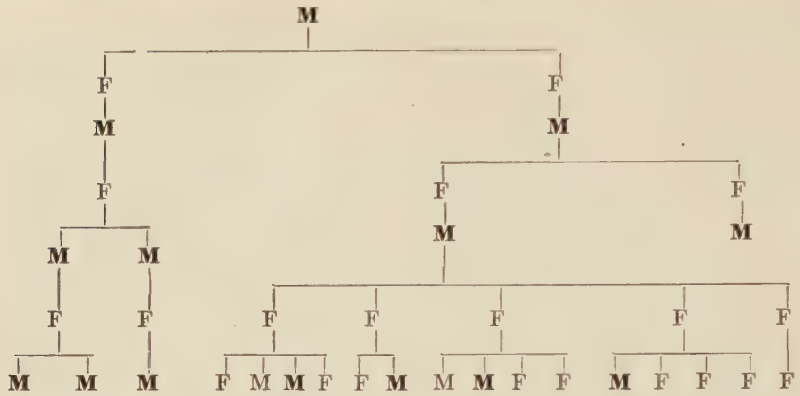
absence of a character or quality—in this instance the absence of a pigment determiner. Two albino parents have only albino children. Normal offspring of an albino and a pigmented parent may transmit the albinic condition.

Albinism is an extreme case of blondeness, all pigment being absent from skin, hair, and eyes. The method of inheritance resembles that of eye color. When both parents lack pigment, all offspring are likewise devoid of pigment. When one parent only is an albino and the other is unrelated, then the children are all pigmented. Whenever pigmented parents have albino children, the proportion of the albinos approaches the ideal and expected Mendelian proportions—25 per cent. Davenport points out that albinos may avoid albinism in their offspring by marrying unrelated pigmented persons. Pigmented persons belonging to albinic strains must avoid marrying cousins, even pigmented ones, because both parents might, in that case, have albinic germ cells and produce one child in four albinic. Albino communities, of which there are several in the United States, are inbred communities, but not all inbred communities contain albinos.

Color-blindness, or Daltonism.—Color-blindness, or daltonism, is a condition probably not localized in the eyes, but due to some defect in the central nervous structure. It is transmitted hereditarily. Color-blindness is much commoner in men than in women. A color-blind man, however, does not transmit color-blindness to his sons, but only to his daughters. The daughters, however, are themselves normal, provided the mother was, yet the daughters transmit color-blindness to half their sons. A color-blind daughter could be produced apparently only by the marriage of a color-blind man with a woman who transmits color-blindness, since the daughter, to be color-blind, must have received this unit character from both parents, whereas the color-blind son receives the character only from his mother; that is, the condition is sex-limited.

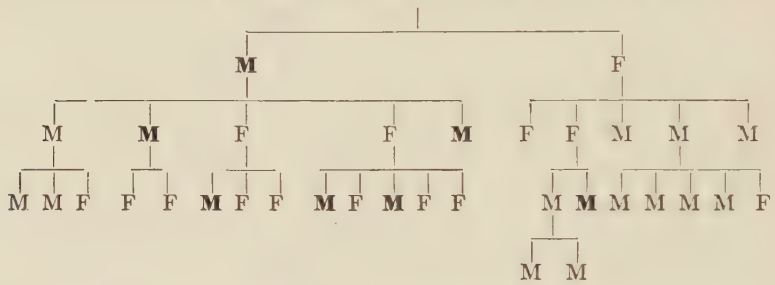
Color-blindness is apparently due to a defect in the germ cell—absence of something normally associated there, with an X-structure which is represented twice in women, once in men.

The following interesting family history, studied by Horner, shows the hereditary persistence of color-blindness and its transmission to male offspring through normal females.

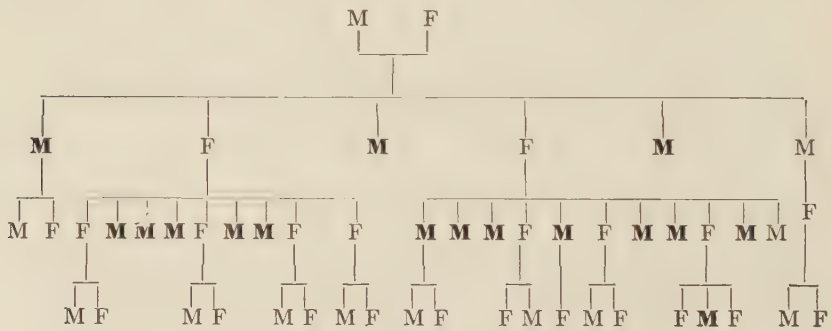


M=Male.
F=Female.
Bold-faced type=Color-blind subjects.

The following pedigree of a family containing color-blind members was worked out by Dr. Rivers among the Todas, an Indian tribe:



Hemophilia.—Hemophilia is a condition in which the blood does not coagulate properly, and those having this condition may bleed to death from a minute wounds. It is transmitted hereditarily and is largely confined to males, although transmitted by normal females. It is one of the best instances of an hereditable character, sex-limited.



(Bold-faced type indicates bleeders.)

The foregoing case, given by Klebbs, is instructive in showing how the tendency, though transmitted through daughters, finds expression only in the males, and in illustrating first a diffusion and then a waning of the peculiarity (Thompson).

Gout.—It is known that gout runs in families, but just what the predisposition is that favors this condition of deranged metabolism is not known. During four centuries one family history showed that out of 535 gouty subjects 309 had a family taint—about 60 per cent. In another family out of 156 cases 140 had a family taint—about 90 per cent. Statistics show that in from 50 to 60 per cent. of all cases the disease existed in the parents or grandparents. It seems clear that some predisposing factor may be transmitted hereditarily, but in any individual case it is not always plain how much is due to heredity and how much to environment.

Brachydactylism.—A typical example of an abnormality is that of brachydactylism, or short-fingeredness, a condition in which each digit comprises only two phalanges—the fingers are all thumbs. This condition seems to be due to an inhibition of the normal growth process, that is, normality implies entire absence of the determiner that stops the growth of the fingers in the brachydactyl. Thus, a brachydactyl person married even to a normal person will beget 100 per cent. or 50 per cent. abnormals, according to circumstances; but two parents who, though derived from brachydactyl strains, altogether lacking the determiner which inhibits the growth of the fingers may have only normal children.

According to Punnett, brachydactylism is a good example of a simple Mendelian case. It behaves as a simple dominant to the normal; that is, it depends upon a factor which the normal does not contain. The recessive normals cannot transmit the affected condition whatever their ancestry. Once free, they always remain free, and can marry other normals with full confidence that none of their children will show the deformity.

Polydactylism.—Polydactylism is a condition in which there are supernumerary fingers or toes. This is a defect which may be transmitted through successive generations.

Myopia.—Myopia can hardly be called a disease in the strict sense, being rather a structural defect in the focusing power of the optical apparatus. It seems that the structural peculiarity which leads to short-sightedness is transmitted.

Cataract.—Bateson and others have collected pedigrees in which cataracts run in families. Presenile cataract especially appears to be transmitted hereditarily.

Retinitis Pigmentosa.—Retinitis pigmentosa is a degenerative disease of the retina which is transmitted hereditarily. Normals may carry

the disease, so that two normal cousins from retinitis stock may have offspring with retinitis. A large percentage of cases of retinitis come from consanguineous marriages.

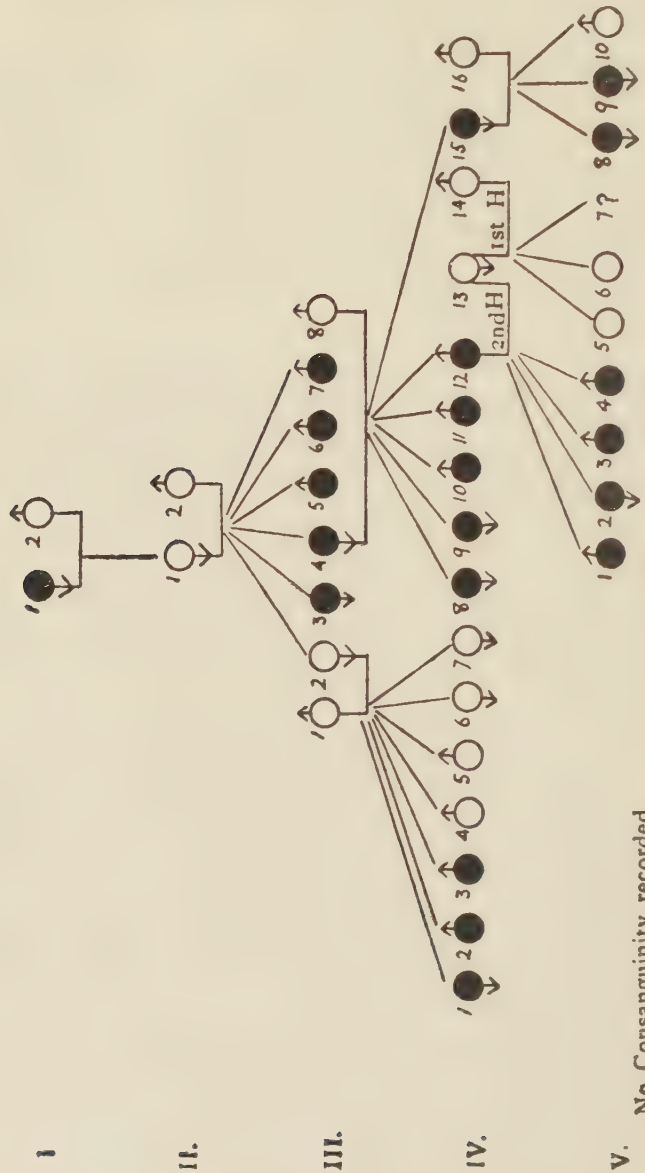


FIG. 65.—FAMILY HISTORY SHOWING POLYDACTYLISM.

Smith and Norwell's case. (From "Treasury of Human Inheritance.")

Diabetes Mellitus.—Hereditary influences seem to play an important rôle in diabetes mellitus, for cases are on record of its occurrence in many members of the same family. Thus, out of 104 cases of dia-

betes mellitus 22 had a family taint—about 20 per cent. Naunyn obtained a history of diabetes in 35 out of 201 private cases, but in only 7 of 157 hospital cases.

Orthostatic Albuminuria.—Orthostatic albuminuria occurs in boys more commonly than girls. These are often the children of neurotic parents, and have well-marked vasomotor instability. Defects or peculiarities in the filtering apparatus in the kidneys may arise as a germinal variation and be handed on from generation to generation. Under conditions which may mean nothing to normal subjects this defect in the kidney may find expression in active disease. In this case, as in gout, it may not be proper to speak of the disease itself being transmitted hereditarily, but the tendency to deviate is so transmitted.

Alcoholism.—It is a common observation that among the offspring of drunkards are many cases of unhealthy, insane, and criminal types. The disastrous results may be manifested by nervous disorders, varying from hyperexcitability to dementia; or as debility and lack of developmental vigor expressed, for instance, in infantilism, want of control, imbecility, or as structural abnormalities, especially of the head and brain. The results are so varied, they suggest that what is inherited is general rather than specific. Thus, the offspring of alcoholic parents are not necessarily predisposed in any one particular direction, except that the nervous system is most liable to be affected. They may be epileptic, idiotic, insane, etc. On the other hand, it is necessary to recognize that what may be inherited is not the result of alcoholism, but rather the predisposition which led the parent to become alcoholic. This is clearly illustrated in cases where the parent did not acquire the alcoholic habit until after the children were born. Clouston observes that "it is not the craving for alcohol that was inherited, but a general psychopathic constitution in which the alcoholic stimulus is an undue stimulus and the mental control deficient." (See page 301.)

Epilepsy.—Brown-Séquard showed conclusively that artificially induced epilepsy in the guinea pig is transmissible. The statistics collected for man give from 9 to over 40 per cent. of cases in which heredity is an important predisposing cause. Gowers gives 35 per cent. for his cases. In the Elwyn cases 32 of the 126 gave a family history of nervous derangement of some sort, either paralysis, epilepsy, marked hysteria, or insanity.

Chronic alcoholism in the parents is also regarded as a potent predisposing factor in the production of epilepsy. Echeherria has analyzed 572 cases bearing upon this point, and divided them into 3 classes, of which 257 cases could be traced directly to alcohol as the cause, 126 cases in which there were associated conditions, such as syphilis and traumatism, 189 cases in which alcoholism was probably the result of

the epilepsy. Figures equally strong are given by Martin, who, in 150 insane epileptics, found 83 with a marked history of paternal intemperance. Of the 126 Elwyn cases in which the family history of this

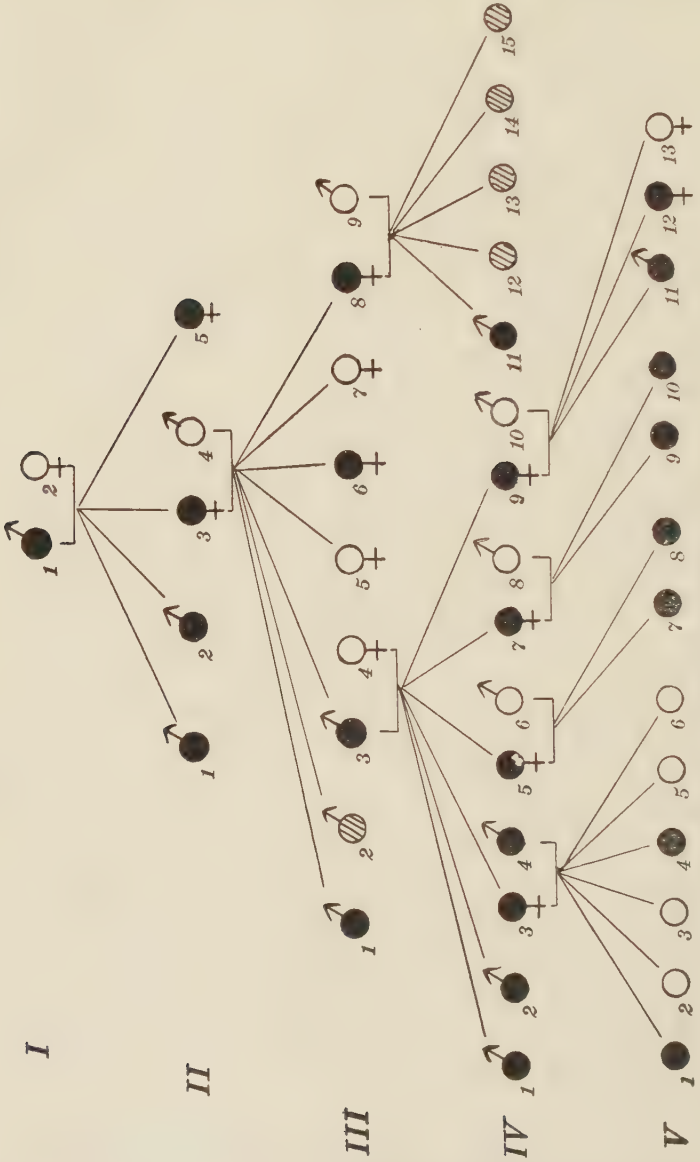


Fig. 66.—Family History Showing Huntington's Chorea.

Last generation incomplete. (Data from Hamilton.)

point was carefully investigated, a definite statement was found in only 4 of the cases (Osler).

Huntington's Chorea.—Huntington's chorea is frequently inherited. The disease is known as chronic hereditary chorea. It was described by

Lyon in 1863, who traced the disease through five generations. Huntington in 1872 gave the three salient points in connection with the disease, viz.: (1) its hereditary nature; (2) association with psychical troubles; and (3) late onset between the thirtieth and fortieth year.

Huntington's chorea is a typical dominant trait. The normal condition is recessive; in other words, the disease is due to some positive determiner. Persons with this dire disease should not have children, but the members of normal branches derived from the affected strain are immune from the disease. This disease forms a striking illustration of the principle that many of the rarer diseases of this country can be traced back to a few foci, even to a single focus; certainly in this case many of the older families with Huntington's chorea trace back to the New Haven colony and its dependencies and subsequent offshoots (Davenport).

Friedreich's Disease—Hereditary Ataxia.—This disease resembles locomotor ataxia, although differing from it in several essential particulars. It begins in childhood and usually occurs in a family having other members of the family affected with the same disease. There are curious forms of incoördination and loss of knee-jerk, early talipes equinus, scoliosis, nystagmus, and scanning speech. The affection lasts for many years and is incurable. In 1861 Friedreich reported six cases of this form of ataxia in one family. Since then it has usually been observed to be a family disease, and is, therefore, assumed to be transmitted hereditarily. The eugenic teaching in this affection, according to Davenport, is that normally all the affected fraternities should marry only outside the strain. Whether all cases of ataxic offspring of one normal parent are derived from consanguineous marriage is still uncertain and warrants hesitation in advising the marriage of any ataxic person.

Imbecility, Defectives, and Delinquents.—Davenport believes that imbecility is due to the absence of some definite simple factor, on account of the simplicity of its method of inheritance. Two imbecile parents, whether related or not, have only imbecile offspring. Davenport states that there is no case on record where two imbecile parents have produced normal children.

Dr. H. H. Goddard, of the Training School for Feeble-Minded, at Vineland, N. J., has studied the ancestry of children in the Vineland institution and has found almost without exception a history of feeblemindedness for several generations. Dr. Goddard's remarkable study of the Kallikak family has already been referred to. In this instance he traced the ancestry of a 22-year-old girl through about 1,100 individuals as far back as the Revolutionary War. Similar studies are being carried out in other institutions and always with similar results.

Insanity.—Insanity is a general term comprising many different

conditions. No general statement can, therefore, be made except that certain forms of insanity are undoubtedly transmitted through succes-

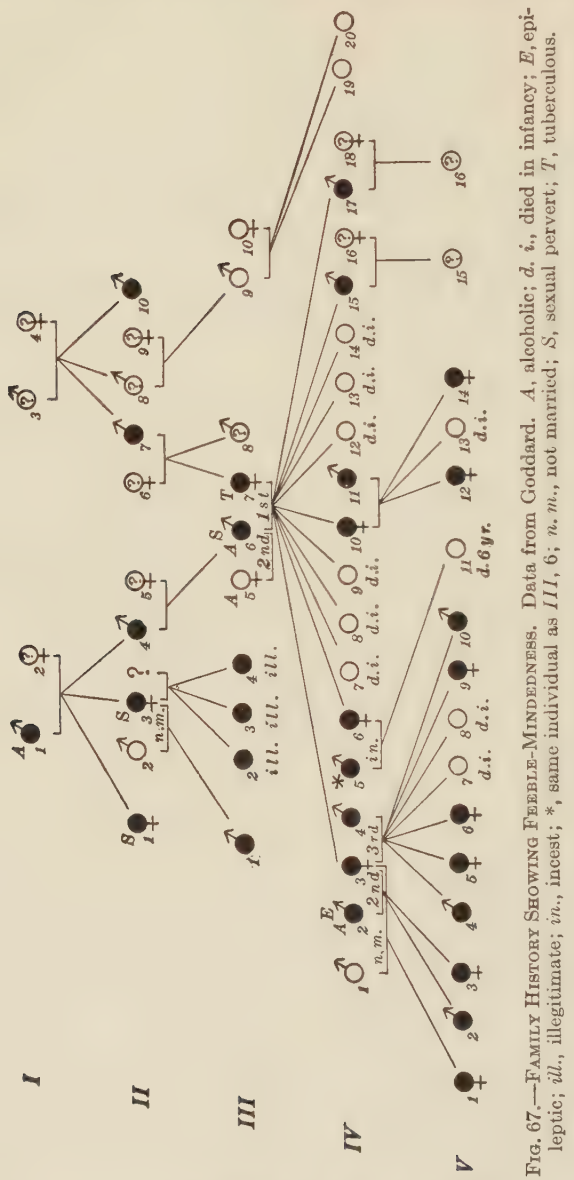


FIG. 67.—FAMILY HISTORY SHOWING FEEBLE-MINDEDNESS. Data from Goddard. *A*, alcoholic; *d. i.*, died in infancy; *E*, epileptic; *ill.*, illegitimate; *in.*, incest; *, same individual as *III*, 6; *n. m.*, not married; *S*, sexual pervert; *T*, tuberculous.

sive generations. Mental diseases are rare in persons free from ancestral taint, except as the result of wounds or toxic influences.

Practically all the statistics accumulated on insanity have limited value to the student of heredity, because they do not give numerical records of the sane members of the families of the insane (see page 298).

REFERENCES

Lock, R. H.: "Variation, Heredity, and Evolution," 1910.

Huxley, T. H.: "Collected Essays," Vol. 2, 1899.

Lamarck, J. B.: "Philosophie Zoologique," 1809.

Darwin, Chas.: "The Origin of Species," 6th Edition, 1872.

Weismann, A.: "Essays Upon Heredity," 1889; "The Variation Theory," 1906; "The German Plasm: A Theory of Heredity" (translated by W. N. Parker and H. Rönnfeld, 1893).

Galton, F.: "Natural Inheritance," 1889; "Hereditary Genius," 1869; "English Men of Science," 1874; "Inquiries Into Human Faculty and Its Development," 1883; "Natural Inheritance," 1889; "Eugenics: Its Definition, Scope, and Aims," 1905.

Davenport: "Statistical Methods," 1904; "Heredity in Relation to Eugenics," 1911.

Pearson, K.: "The Grammar of Science," 2d Edition, 1900.

Bateson, W.: "Materials for the Study of Variation," 1894; "Mendel's Principles of Heredity," 1909.

DeVries, H.: "Die Mutationstheorie," 1901; "Species and Varieties: Their Origin by Mutation," 1905.

Wilson, E. B.: "The Cell in Development and Inheritance," 2d Edition, 1900.

Thompson, J. A.: "Heredity," 1908.

Castle, William E.: "Heredity," 1911.

The Proceedings of the Roy. Soc. of Medicine, 1909, Vol. II: "The Influence of Heredity on Disease," with Special Reference to Tuberculosis, Cancer, and Diseases of the Nervous System.

Punnett, R. C.: "Mendelism," 1911.

SECTION III

FOODS

CHAPTER I

GENERAL CONSIDERATIONS

Foodstuffs fall naturally into two great divisions: (1) those derived from the animal kingdom, and (2) from the plant kingdom. The animal foods are much more apt to convey infections or to possess injurious properties than foods derived from plant life. Of the animal foods meat and milk are the chief offenders. Water ordinarily is not classed as a food, and is discussed in a separate chapter.

The increase of food poisoning and the increase of diseases caused by infected foods are more apparent than real. The subject is better understood, and cases are now recognized and reported that were formerly misinterpreted. The hygienic conscience of the people has been aroused, and a demand has been established for clean, fresh, wholesome foodstuffs. The separation of the producer and the consumer and the demands of large cities have made these sanitary reforms eminently necessary. The pure food laws, the meat inspection act, the milk ordinances, and the local surveillance over markets, provision shops, dairies, etc., are all part of the general movement to obtain a reasonably decent and safe food supply.

People should be educated to demand flesh from healthy animals, cut up and handled in a careful manner by butchers free from disease, and to demand garden truck grown in clean dirt and not in soil polluted with human excrement. Food must be guarded in transportation and purveyed in markets and shops so as to be protected from flies, rats, dust, and unnecessary human contact.

The prophylactic and therapeutic uses of food are growing subjects. It is only necessary to point out the importance of diet in the prevention and treatment of tuberculosis, diabetes, nephritis, arteriosclerosis, gout, rheumatic affections, disorders of metabolism, dyspepsia, gastric ulcer, infantile diarrheas, and many other affections. The proper amount and quality of food is one of our important preventive measures.

Food may affect health in a great variety of ways:

(a) Foods may be naturally poisonous, as in the case of certain mushrooms, some fish, or the alkaloids in various species of plants.

(b) Poisonous substances may develop in the food as a result of bacterial activity, as sausage poisoning (botulism). In this class are also included the so-called ptomains or putrefactive poisons.

(c) Foods may convey foreign or accidental poisons. This class includes mostly the metallic poisons and chemicals added as adulterants, as lead, copper, arsenic, formaldehyde, sulphites, etc.

(d) Foods may contain animal parasites, such as trichina and tapeworms. These infections, as a rule, occur as ante-mortem infections in the animal. Plant foods may carry the eggs or larvæ of various animal parasites.

(e) Foods may contain vegetable parasites. Both animal and vegetable foods may convey bacteria pathogenic for man. The harmful varieties are more often found upon animal foods than upon vegetable foods. The food animals may be infected before death, or the meat may become infected while it is being cut up or handled. The best example in this class is paratyphoid infection, sometimes called meat poisoning; also typhoid bacilli in oysters, or on celery, etc.

(f) Foods may contain special poisons, as, for example, solanin in sprouted potatoes, or ergot in rye.

In this class are also included spoiled corn and its relation to pellagra.

(g) Food may be injurious as a result of abnormality of amount or composition of diet. Thus, an excess of food predisposes to obesity and perhaps to arteriosclerosis and diseases of the liver and kidneys. An insufficient amount undermines health. A monotonous diet, especially of polished rice, leads to beri-beri; lack of organic acids induces scurvy; defective alimentation, especially a deficiency of lime salts, predisposes to rickets. Highly spiced and stimulating diets are irritating both to the digestive tube and the organs of excretion. An excess of fats produces a condition resembling acidosis, particularly in children. An unbalanced ration long continued is apt to be harmful. Thus, an excess of protein induces putrefactive changes with its dangers of autointoxication. Eating when fatigued, or improper mastication, are causes of indigestion. Drinking too little water is a common dietetic error.

(h) Finally, foods may not be poisonous in themselves, but may be harmful to persons who lack ability to digest them or lack the mechanism by which they may be assimilated. Thus, certain forms of protein habitually produce symptoms resembling anaphylaxis in persons who are sensitized. This occurs most commonly with sea food, but also takes place with strawberries, eggs, and other forms of protein.

THE USES OF FOOD

The two ultimate uses of all food are to supply the body, (1) with materials for growth or renewal, and (2) with energy or the capacity for doing work. The potential energy received in a latent form stored in the various chemical combinations in foods is liberated as kinetic or active energy in two chief forms, heat and motion. Force is the manifestation of energy, and the force developed by a healthy man may be measured in foot pounds. A foot pound is the amount of energy expended or force required to lift mechanically a weight of one pound to a height of one foot.

The work of an average man is calculated at about 2,000,000 foot pounds per diem (R. H. Thurston). This may exceptionally be increased to 3,000,000 foot pounds. Ordinarily less than one-fifth of the total energy of the body is expended in motion, and more than four-fifths in heat production.

The total intake of energy into the body is derived from food plus the oxygen of the inspired air. The total output of energy is computed from: (1) the heat of combustion of the unoxidized ingredients of the urine and feces; (2) the energy liberated as body heat, and (3) the energy of external muscular work, or the work of the voluntary muscles (Thompson).

Whether alimentary substances are burned outside of the body or oxidized within the body, the resulting waste products are similar. No substance is a good food unless it fulfills two conditions, viz.: easy assimilation and complete combustion.[1]

Two methods may be employed to study the energy-producing power of food in the body: (1) a careful and prolonged study of subjects who are allowed to follow their usual vocations, but whose food and excreta are carefully measured and analyzed; (2) the shorter method of enclosing a man for a brief period, not exceeding a few days, in a cabinet known as a calorimeter.

The unit of measurement is the calorie, which is the amount of heat required to raise one kilogram of water from 0° to 1° C. This equals 3,100 foot pounds, or approximately the heat required to raise the temperature of one pound of water 4° F. Fuel value is a term denoting the total number of calories derived from a gram or pound of any given food substance if it is completely combusted within the body. The fuel values are calculated for a given food by the factors of Rubner as follows:

[1] It is not sufficient to know merely the amount and caloric value of the coal fed to a furnace, and subtract therefrom the amount of unconsumed ash. We must know how much of the heat generated has been *utilized*.

4.5 calories per gram of either protein or carbohydrate.
9.3 calories per gram of fat.

Atwater and Bryant compute the food factors as 4 calories per gram for proteins and carbohydrates and 8.9 for fats, in a mixed diet. C. F. Langworthy gives the fuel value of the three chief classes of nutrients as follows:

1 pound of protein yields	1,860 calories
1 pound of fats	4,220 calories
1 pound of carbohydrates	1,860 calories

From a chemical standpoint foods are oxidized or burned to simpler compounds during the process of digestion and metabolism within the body. Food is, therefore, fuel. The oxygen to feed the flame is mainly furnished by the inspired air, hence active breathing of pure fresh air is essential and one of the best stimuli for complete metabolism. It is the common experience of all persons that digestion and the utilization of foods are favorably promoted by life in the open air.

CLASSIFICATION OF FOODS

Foods may be classed in various ways. Thompson divides them into four groups, according to (1) their physical properties, (2) their source, (3) their composition, and (4) their function, or the rôle which they perform in the animal body.

Physical Properties.—Foods are classed in accordance with their general physical properties first into solid, semisolid, and liquid foods; secondly, into fibrous, gelatinous, starchy, oleaginous, crystalline, and albuminous foods. Foods are also classed as foods, beverages, and condiments.

Sources.—Foods may be classed as to their source primarily into (a) animal and (b) vegetable foods.

Animal foods consist of meat, fowl, fish, shellfish, crustaceans, insects and their products (honey), eggs, milk and its products, animal fats, gelatin.

The vegetable foods are subdivided into cereals, vegetables proper, fruits, sugar, gums, vegetable oils and fats.

Composition and Function.—The simplest chemical classification possible is that advocated by Liebig, who was the first to suggest a really scientific definition of foods. He grouped all foods into two classes: nitrogenous and non-nitrogenous. Each of these classes contains food materials from both the animal and vegetable kingdoms, although the majority of the animal substances belong to the nitroge-

nous and the majority of the vegetable substances to the non-nitrogenous group.

Nitrogenous foods contain proteins and include gelatinoids and albuminoids, substances which resemble albumin. They consist chiefly of the four elements: carbon, oxygen, hydrogen, and nitrogen, to which a small proportion of sulphur and phosphorus is usually joined. The nitrogenous foods were regarded by Liebig as containing plastic elements; that is, they are essentially tissue builders or flesh formers. The non-nitrogenous group Liebig called respiratory or calorifacient foods, because their function in the body is largely to furnish fuel to maintain animal heat. It is now known that the non-nitrogenous foods supply energy for muscular action, hence they are also called force producers, to distinguish them from the nitrogenous or tissue builders. This is a convenient distinction, but it must not be held too absolutely, for in certain conditions the tissue builders are used as force and heat producers as well.

Foods are now ordinarily classed as: (1) nitrogenous; (2) starchy; (3) oily, and (4) condimental. Examples of nitrogenous foods are lean meat, the white of eggs, or the casein in milk. The gluten of wheat and the zein of corn are also typical nitrogeneous constituents. Peas and beans contain large percentages of nitrogenous matter. The nitrogenous or protein substances build and repair tissue, and to a less extent serve as fuel to yield energy in the forms of heat and muscular power.

The starchy or carbohydrate foods are represented by the cereals, the tubers, such as potatoes, the sugars of the cane, beet, fruits, etc., and glycogen in flesh.

Fats or oily foods are represented by butter, olive oil, cotton-seed and other oils, the fat of meat, the oil of nuts and seeds. All vegetables contain more or less oily substances. The fats as well as the carbohydrates serve as fuel to yield energy in the form of heat and muscular power.

Mineral matter or ash performs an important service in forming bone and assisting in digestion and metabolism. These substances are ordinarily not classed as foods; however, life cannot be maintained without them.

Among the condiments are classed: spices, such as pepper, mustard, cinnamon, cloves, etc.; also coffee, tea, and alcoholic beverages.

THE AMOUNT OF FOOD

Excessive Amounts.—The amount of food required varies greatly with conditions. In civilized communities, where cooking is a fine art, the number and variety of food preparations are so great that the ap-

petite is often stimulated beyond the requirements of the system, and consequently more food is eaten than is necessary or desirable to maintain the best bodily health and vigor. Gluttony results in overdevelopment and overwork of the digestive apparatus; the stomach and bowels become enlarged; the liver is engorged, and a predisposition is established to degenerative changes, fatty heart, etc. The quantity of food required to maintain the body in vigor varies with the climate and season, clothing, occupation, work, and exercise, the state of individual health, age, sex, and body weight.

Both overeating and overdrinking may be temporary or chronic. When chronic it may lead to such diseases or diatheses as obesity, gout, lithemia, oxaluria, or the formation of renal, vesical, and hepatic calculi. It is very certain to cause congestion of the liver and the condition known as "biliousness," in which the stomach and intestines are engorged, constipation results, the tongue is heavily coated, the bodily secretions are altered in composition, the urine especially becomes overloaded with salts, the liver becomes congested, and, finally, the nervous and muscular systems are affected, which result in the production of headache and feelings of fatigue, lassitude, drowsiness, and mental stupor.

Insufficient Food.—Starvation or asitia is a term which technically applies to the lack of sufficient food for the maintenance of the body, while inanition means the lack of the assimilation of food by the tissues. When food is completely withheld, life cannot be prolonged beyond six or ten days in the majority of instances. Professional fasters have gone 41 days without anything but water. If food is withheld suddenly, the sensation of hunger gradually increases, becomes extreme, lasts for two or three days, and slowly disappears. It is accompanied by a gnawing pain in the epigastrium, which is relieved on pressure. The pain may disappear, but it is followed by a sensation of extreme weakness or faintness, which is both local in the stomach and general throughout the body. Even though the pain disappears, the sensation of hunger may occasionally reassert itself, when all food is withheld, until death, or until the subject becomes insane or unconscious.

Hunger is not always a reliable guide to the need of the system for food. Some dyspeptics are always hungry and eat more than they can digest. A habit of rapid eating does not satisfy the sensation of hunger. More food may be taken than is necessary, because it has not had time to meet the needs of the system before the meal is over. Cannon has shown that the sensations of hunger come and go rhythmically, appearing synchronously with the contractions of the empty stomach.

The statement is frequently made that, when starvation occurs upon a large scale, affecting a community with famine, pestilence is sure to accompany it. Thus, disease has often been rampant in Ireland when

the potatoes have failed, and in India when the grain supply has given out. Much of the illness which occurred in the early history of the Crimea was coincident with insufficient food, and it is stated that in the middle ages the ravages of pestilential diseases, such as typhus, smallpox, plague, etc., were always worse in times of general starvation. The history of epochs of famine in siege or otherwise is always accompanied by outbreaks of violence, for hunger begets ill temper, vice, and crime. This has occurred of late years, notably in Athens, Florence, and London, and in Paris during the Commune. There is, however, no very definite relationship between famine and epidemics. The depressed vitality caused by insufficient food does not account for epidemics of plague, smallpox, relapsing fever, typhus fever, and other pestilential diseases, sometimes called famine fevers. The reasons for this have been discussed under Immunity.

Unbalanced Diets.—Unbalanced diet may produce anemia from lack of meat or other food; scurvy from lack of fresh fruits and vegetables, with preponderance of salty meat and fish; rickets and marasmus from an excess of amylaceous and lack of animal food, necessary salts, etc.; a form of acidosis from too much fat, especially in babies; acne or eczema from food too rich in carbohydrates or fats; constipation from too nutritious and concentrated diet; and gout from various dietetic errors. Unbalanced diets are responsible for a long list of affections, a type of which is beri-beri, caused by a monotonous diet consisting chiefly of unpolished rice.

Salts in the Diet.—Common organic or vegetable acids, such as citric from lemons and oranges, tartaric from grapes, malic from apples, etc., usually exist in combination with the bases, calcium, sodium, potassium, etc., when derived from fresh vegetables and fruits. They are indispensable articles of food, for when absorbed they form carbonates, which aid in maintaining the alkalinity of the blood. Prolonged deprivation of them may result in scurvy. Lack of sufficient potash salts, especially potassium carbonate and chlorid, is also a factor in producing scurvy, and the condition is intensified by the excessive use of common salt.

If calcium phosphate is deficient in the food of the young, growing infant, the bones are poorly developed and so soft that they yield to the strain of the weight of the body and become bent, as occurs in rickets.

Lack of inorganic salts in the food impoverishes the coloring matter of the red blood corpuscles on which they depend for their power of carrying oxygen to the tissues, and anemia and other disorders result. An ash-free diet soon causes serious symptoms.

Longworthy gives the following as the estimated amount of mineral matter required per man per day:

Phosphoric acid (P_2O_5)	3	to 5	grams
Sulphuric acid (SO_3)	2	to 3.5	"
Potassium oxid	2	to 3	"
Sodium oxid	4	to 6	"
Calcium oxid	0.7	to 1.0	"
Magnesium oxid	0.3	to 0.5	"
Iron	0.006	to 0.012	"
Chlorid	6	to 8	"

ADULTERATION OF FOOD

Adulteration of food consists of a large number of practices, some of which are fraudulent, others technical in nature. Some forms of adulteration are injurious to health, but for the most part they have an economic rather than a sanitary significance. Foods may be adulterated in a variety of ways: by the removal of nutritive substances; by the addition of injurious substances; by the fraudulent substitution of cheaper articles; by misbranding; or by the sale of food that is filthy, decomposed, or putrid.

Prior to the passage of the Pure Food and Drugs Act in 1906 a very large percentage of the food sold in the United States was found to be adulterated in one way or another. Thus, at the Agricultural Experiment Station in Kentucky 40 per cent. of 727 samples were adulterated; at the Connecticut Agricultural Experiment Station 41.5 per cent. of 574 samples of spices were found adulterated, and over 25 per cent. of coffee samples were adulterated (1899).[1]

Among the common adulterations may be mentioned the following: cotton-seed oil is sold as olive oil; honey may contain glucose; cocoa and chocolate are frequently adulterated with both starch and sugar; coffee is extensively adulterated with caramel, pea-meal, chickory, and saccharose extracts; lard is mixed with cheaper fats or cotton-seed oil; saccharin is substituted for cane sugar; cereals give bulk and weight to sausages; gypsum or bran is added to flour; barium sulphate to powdered sugar, flour or turmeric or corn meal to mustard. Oleomargarin is sold as butter; distilled and colored vinegar is sold as cider vinegar; ground spices are adulterated with cocoanut shells, rice, flour, and ashes; water, sugar, and tartaric acid is sold as lemonade;

[1] In Massachusetts the State Board of Health began to examine foods for adulteration in 1883. It was then found that between 60 and 70 per cent. of all foods examined were adulterated. As a result of official surveillance the percentages fell in a few years to, approximately, 15 per cent. and have remained between 10 and 20 per cent. since. This does not mean that from 10 to 20 per cent. of all foods found on the market are adulterated, for, to a great extent, samples are collected from suspicious sources, so that the ratio of adulteration of foods analyzed in the laboratory is higher than that of the same foods sold on the market.

wines and liquors are sometimes adulterated with alum, baryta, caustic lime, salicylic acid, and hematoxylin. Terra alba, kaolin, and various pigments are sometimes added to candies; gum drops are largely made with petroleum paraffin products; much of the maple sugar formerly sold was made from glucose and coloring matters.

A food is considered adulterated in accordance with the Food and Drugs Act of June 30, 1906: (1) "If any substance has been mixed and packed with it so as to reduce or lower or injuriously affect its quality or strength." This is the simplest form of adulteration, and a good example is the addition of water to milk. Cocoa shells are sometimes mixed with cocoa or chocolate. Glucose and caramel are added to maple sugar; talc to flour.

(2) "If any substance has been substituted wholly or in part for the article." As illustrations we have the substitution of cotton-seed oil or corn oil for olive oil; glucose or saccharin for sugar; cereals, which cost about five cents a pound, for meat, which averages fifteen cents a pound, in sausage. Apple cores and parings are frequently used as a substitute for currants and other fruits in jellies.

Saccharin is several hundred times sweeter than sugar and comparatively cheap. It has, therefore, been used as a substitute for sugar as a sweetening agent in the inferior qualities of ginger ale, and to some extent in canned corn, peas, etc., as well as in candies and other articles. Saccharin is a chemical obtained from coal tar and is without food value; it is not entirely harmless. The Referee Board reports that "the continued use of saccharin for a long time in quantities over 0.3 of a gram per day is liable to impair digestion; and the addition of saccharin as a substitute for cane sugar reduces the food value of the sweetened product and hence lowers its quality." Saccharin-containing foods are therefore regarded as adulterated within the meaning of the Food and Drugs Act.

(3) "If any valuable constituent of the article has been wholly or in part abstracted." Skimming milk is a good illustration of this part of the law, or the abstraction of cocoa butter from chocolate. There is, however, no objection to abstracting valuable or nutritive substances provided the label properly announces the facts; thus, skimmed milk or cocoa are legitimate foods. So also the caffein may be taken out of coffee and sold as caffein-free coffee. The essential oils are sometimes extracted from cloves or other spices, which are subsequently ground and used as an adulterant with unextracted spice.

(4) "If it is mixed, colored, powdered, coated, or stained in any manner whereby damage or inferiority is concealed." This is a very frequent form of adulteration, and, as a rule, is undesirable and sometimes injurious. Substances used to color foods are usually considered in three classes: (1) mineral dyes, (2) vegetable dyes, (3) anilin or

coal-tar dyes. The principal mineral dyes are: copper sulphate, oxid of iron, and potassium nitrate. Copper sulphate is used to give a green color to peas, pickles, and similar foods. The copper probably unites with the albuminous matter to form new compounds which have a bright green color. The oxid of iron and also sulphites are used upon meat to give it a red color; potassium nitrate will also give a bright red color to meat. Many vegetable dyes are used, such as annato (the juice of the *Bixa orellana*, a South American tree), which is used to color butter. Carrot juice is also used; turmeric in mustard; and logwood in wines. The coal-tar dyes have largely replaced the vegetable and mineral pigments in foods, on account of their brilliant color and cheapness. They are used in sausages, confectionery, jellies and jams, meats, flavoring extracts, etc. The artificial coloring of food is a false standard and serves no useful purpose. When the coloring matter is used to conceal damage or inferiority the practice is indefensible, as when spoiled meats are made to look bright red and fresh, or when oleomargarin is colored in order to imitate butter and sold as such. Flour may be bleached with nitrogen peroxid, thus giving an inferior grade the appearance of first quality flour. The NO_2 is produced by electric action and nitrites in appreciable quantities remain in the flour. Fruits are bleached by exposure to sulphur fumes, which leaves objectionable sulphur compounds. Candies and chocolate are often coated with gum bezoin or shellac.

(5) "If it contains any poisonous or other added deleterious ingredient which may render such article injurious to health." This section of the law is intended to include adulterants, such as formaldehyde, sulphites, arsenic, hydrofluoric acid, lead, salicylic acid, borax and boracic acid, as well as any other injurious substance. Most of the storm center of the opposition to the Pure Food Law is centered around this paragraph, owing to the difficulty of deciding in certain instances whether small amounts of benzoic acid or benzoates, boric acid or borates, are injurious to health or not. These substances are discussed more in detail under chemical preservatives.

(6) "If it consists in whole or in part of a filthy, decomposed, or putrid animal or vegetable substance or any portion of an animal unfit for food, whether manufactured or not, or if it is the product of a diseased animal or one that has died otherwise than by slaughter." Examples: oysters contaminated with sewage; eggs known as "rots and spots"; animals which have died otherwise than by slaughter; figs containing an excessive quantity of worms and worm excrement. This paragraph of the law has caused much discussion, especially the meaning of the word "decomposed." This question is considered more in detail under the paragraph "Decomposed Foods."

Misbranding.—The term "misbranding" is specifically defined in

the Food and Drugs Act and provides for all possible conditions of fraud, mislabeling, imitation, substitution, and other forms of deception. Misbranding is regarded as a form of adulteration under the Food and Drugs Act. The practices of misbranding under any circumstances are so evidently fraudulent or dishonest that they cannot be justified on any score and are wholly condemned. It is true that many instances of misbranding do not directly affect health, except in so far as they deceive the consumer; that is, he is purchasing at a high price an article which contains less nutritive value than claimed for it. An honest label which correctly states the character, origin, amount, and the constituent parts of an article is as much a desideratum in food products as it is in commercial articles of all kinds. Honest labeling is the heart and soul of the pure food movement.

DECOMPOSED FOODS

Decomposition is defined as natural decay. In this sense all organic substances, both animal and vegetable, living or dead, are decomposed, for decomposition and recomposition occur as a constant feature of life's processes. At the moment of death recomposition ceases, while decomposition continues. In one sense the hardest rocks decompose or disintegrate; bicarbonate of soda decomposes in the presence of an acid, and many substances decompose in the presence of oxygen, especially when heated. In other words, while decomposition is usually the result of bacterial activity in organic substances, it may also take place as the result of physical, chemical, or electrical agencies. The word "decomposition" is not used in this technical sense in the Pure Food and Drugs Act; there it has the meaning of the word used in ordinary, every-day parlance. Just where technical decomposition ceases and objectionable decomposition begins is often difficult to determine. Decomposition may be objectionable either to the senses or to health. We purposely permit many of our foods to decompose before they are used. Thus, meats hang three days or longer in order to render them more tender and to improve their flavors. During this time decomposition takes place with the production of acids. Some persons prefer meats when highly decomposed or gamy. Bread, cheese, butter, buttermilk, sauerkraut, vinegar, cider, and many other foods are products of decomposition. The line must, therefore, be drawn between decomposition that is objectionable and decomposition that is technical. It is difficult to draw the line at decomposition that is objectionable to the senses, for a cheese regarded as a delicacy by one person may be highly objectionable to another. The principal point, then, for consideration is the decomposition that is harmful to health.

Fermentation and Putrefaction.—The question is further complicated when we consider that there are very many kinds of decomposition. Two main groups are recognized: (1) fermentative decomposition, and (2) putrefactive decomposition. Even the Pure Food Law distinguishes between foods that are decomposed and foods that are putrid. Fermentative decomposition refers to the breaking down of carbohydrates with the formation of acids (lactic, acetic, butyric), alcohol, carbon dioxid, etc. Putrefactive decomposition refers to the breaking down of nitrogenous substances, usually with the production of alkalinity. The end products of putrefaction are ammonia, nitrates, carbon dioxid, etc., all simple, stable, inorganic compounds which, in ordinary concentration, are not poisonous. It is then the intermediate cleavage products of putrefaction and the end products of fermentation that may be poisonous. The question of decomposition is still further complicated by the fact that there are very many different kinds of fermentation and of putrefaction. Each particular microorganism breaks down organic matter in a specific and limited sense. Ordinarily these processes result from a combination of bacteria or symbiosis, in which aerobic and anaerobic organisms each play a part. As a rule, putrefaction does not take place in the presence of fermentation. In this sense carbohydrates protect nitrogenous matter; further, the products of fermentation are much less poisonous than some of the products of putrefaction.

"Ptomain" Poisoning.—Ptomains are secondary cleavage products of protein putrefaction. Vaughan defines a ptomain as an organic chemical compound, basic in character, and formed by the action of bacteria on nitrogenous matter. On account of their basic properties ptomains resemble the vegetable alkaloids and are, therefore, called putrefactive alkaloids. They are sometimes called "animal" alkaloids, but this is a misnomer, for they also are formed in the putrefaction of vegetable matter.

The term "leukomain" is used to cover the same or similar basic substances which result from tissue metabolism within the body; that is, leukomains are produced in the living body, ptomains in dead organic matter.

The great majority of ptomains are not poisonous or less toxic than the corresponding ammonia compound. Ptomains include substances which are chemically very different. The classification is not a scientific one, and is gradually being abandoned. In fact, most cases of so-called ptomain poisoning are really infections with microorganisms belonging to the paratyphoid group.

The products produced during the various stages of protein putrefaction resemble the chemical products produced during the stages of protein digestion. Putrid, or putrefying, organic matter is not neces-

sarily poisonous or even harmful. There is a time when decomposing meat or cheese or other nitrogenous substance reaches the height of its toxicity, then gradually declines, and finally becomes inactive. The terminal products of putrefaction, or even the later stages, while highly offensive to the taste and smell, may be quite harmless. Therefore, cheese, if toxic, is most poisonous when green; that is, during the intermediate stages. Meat, if toxic, is most poisonous from the fourth to the eleventh day of putrefaction. The poisonous properties of other foodstuffs have a similar relation to the stage of putrefaction.[1]

Chemically, ptomains are ammonia substitution compounds; two-thirds of them contain only carbon, hydrogen, and nitrogen. Those having oxygen in their composition are the more poisonous. Most ptomains are inert or are no more poisonous than the corresponding ammonia salts. In composition they show a predominance of the amin radicle (NH_2). Of the bases containing oxygen, most of them are trimethylamins [$(CH_3)_3N$]. It was Brieger who pointed out that a certain quantity of oxygen is necessary for the formation of poisonous bases. These poisonous bases appear about the seventh day of putrefaction and then disappear.

The decomposition products of putrefaction have long been studied by chemists. All the studies made before bacterial activity was understood are now only of historical interest. The distinguished physician, Panum, was the first to demonstrate positively the chemical nature of the poisons formed in putrid flesh. He obtained an aqueous extract which retained its poisonous properties after boiling eleven hours. Panum studied these poisonous substances by intravenous injections upon dogs. These observations have been abundantly confirmed, but so far it is doubtful whether anyone has succeeded in isolating the poisonous substances in a pure state.

In 1886 Bergmann and Schmiederberg obtained sepsin from putrid flesh and from decomposing bodies. This substance was obtained in needle-shaped crystals, and small doses injected into dogs caused vomiting and bloody diarrhea. It was then believed that the putrid poison of Panum had been isolated and was identical with sepsin. Further investigation, however, showed that this was not the fact. Selmi, an Italian, added valuable information to the study of this question, and, what is probably more important, gave an impetus to the study of the chemistry of putrefactive changes. Selmi was the first to suggest the name ptomain; he showed that there are a great number of alkaloid-like substances among the products of putrefaction. Some of these

[1] It seems that advanced decomposition favors the destruction of the poisonous substances formed earlier in the process; it is further known that most of the pathologic microorganisms die during active fermentative or putrefactive changes.

may be extracted with ether from an alkaline solution, some with ether from an acid solution, some with chloroform from either an acid or alkaline solution, and some with amylic alcohol, and after all these extractions there yet remain alkaloidal bodies in the putrid infusion. This gives us an indication of the great number of ptomains. We long remained ignorant of the chemistry of these substances until Nencki in 1876 made the first ultimate analysis and determined the empiric formula for ptomains. In 1871 Lombroso showed that an extract from moldy corn produced tetanic convulsions in animals. This observation was part of Lombroso's long struggle to discover the chemical substance responsible for pellagra. In 1885 Vaughan detected in poisonous cheese an active agent to which he gave the name tyrotoxicon. However, Vaughan afterward admitted that this is not the substance most commonly found in poisonous cheese, though the names tyrotoxicon poisoning and ptomain poisoning remain in popular parlance. Brieger kept the name ptomain, but applied it to those basic substances only that were produced in the life processes of bacteria. He classed under the head of ptomains a great number of poisons which he succeeded in isolating from putrid flesh, decomposed fish, rotten cheese, decomposed glue, cadavers in various stages of decomposition, from poisonous mussels, etc. Since these a long list of basic substances or ptomains have been described.[1] As before pointed out, the great majority of them are not poisonous, or slightly so. The list includes a variety of substances which are chemically very different. The classification is not a scientific one, and is gradually being abandoned. Even at the time of Brieger's investigations it was found necessary to distinguish between poisonous and non-poisonous ptomains, and on this account the term toxine was adopted—a distinguishing name for poisonous products of bacterial activity.

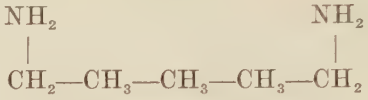
The best known poison which has been isolated in an approximately pure state from decomposing nitrogenous material is sepsin. Much work has been done upon this substance by Schmiederberg and recently by Faust, who obtained sepsin in a purified state in sufficient quantities carefully to study its action and composition. Faust obtained the crystals from putrefied yeast and blood; 25 milligrams of the sulphate introduced intravenously will kill a large dog in two hours.

Sepsin has the following chemical structure:

Dihydroxypentamethylenediamin.

[1] See Vaughan and Novy: "Cellular Toxins."

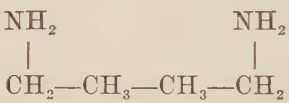
Sepsin is very unstable. It is rendered inactive at 60° C. for a short time, and is readily converted into cadaverin or pentamethylenediamin. The chemical structure of cadaverin is:



Pentamethylenediamin.

Cadaverin is one of the best known of the ptomain group. Its presence indicates that the putrefactive process at one time contained sepsin which, by reduction, has been changed into cadaverin.

Putrescin is another diamin, which almost invariably occurs together with cadaverin, to which it is closely related. It was first described by Brieger in 1885, and has been obtained from putrefying internal numan organs, herring, mussels, etc. It is recognizable on the fourth day of putrefaction, and appreciable quantities appear by the eleventh day. It is still present after two or three weeks. Baummann in 1888 showed the rational formula to be



Tetramethylenediamin.

Putrescin is a homolog of cadaverin and appears in putrefaction before that substance.

The more the question of ptomains is studied the less do they appear concerned in cases of food poisons. It is now clear that most, if not all, cases of so-called ptomain poisoning are nothing more nor less than acute infections with *B. paratyphosus, B. enteritidis, B. choleræ suis,* and other microorganisms belonging to this group. A number of bacteria ordinarily harmless are capable, under certain conditions, or in overwhelming numbers, of producing acute gastrointestinal disturbances. Some of these microorganisms and their effects are discussed more particularly under Meat Poisoning.

Less is known concerning the decomposition of the fats. It is quite possible that some of these substances may be exceedingly poisonous. Thus, while cholin in itself is not very toxic, Hunt has shown that acetyl-cholin is one hundred thousand times more poisonous. Cholin is a base widely distributed in nature; it is found in the yolk of eggs, in bile, brain substance, fat, seeds, and other substances. It can also be prepared from pure lecithin, which is a fatty body normally pres-

ent in brain substance, yolk of eggs, and perhaps all cells. Milk, for example, contains about one per cent. of lecithin. The lecithin may be readily decomposed by bacterial action perhaps to cholin and cholin salts. While acetyl-cholin has never been demonstrated in milk, it is possible that this or similar poisons may be produced in decomposing foodstuffs.

PRESERVATION OF FOODS

The preservation of meat, milk, vegetables, and other perishable foods is one of the most important questions we have to deal with in the whole range of hygiene. Fermented and decayed foods must be looked upon with suspicion. The proper preservation of foodstuffs involves not only the art of keeping them "fresh" and wholesome, but also keeping them so that they will not lose their nutritive value. Finally, foodstuffs must be preserved so that they will not acquire injurious properties. The preservatives ordinarily in use are: cold, drying, salting, smoking, canning, preserving, and chemical treatment.

Practically all these methods have long been in use. The only modern innovation in the preservation of foods is in the perfection of the old processes, based upon our knowledge of antiseptics and germicides. Heat and cold represent old family methods which have been extended and improved in the modern canning and cold storage industries. The drying of fruits, fish, and meats is a practice of very ancient origin. The use of salt doubtless antedates all historical records. Sugar either alone or with acetic acid in the form of vinegar and with various spices is an old contrivance and well known everywhere. The application of creasote obtained crudely from the smoke of incompletely burned wood is the ancient forerunner of some of the modern packing processes.

Concerning the value and legitimacy of these old family methods there is comparatively little difference of opinion; salt meat is not as good as the fresh article; dry apples do not make the best apple pie; chipped beef is not an adequate substitute for a fresh steak. However, it is absolutely necessary to preserve food in some way in order to tide over the winter or the dry seasons, to furnish food to people living and working in desert and arid regions, and to feed the hordes of people massed together in great cities. It would be impossible to maintain the large population of a modern metropolis if it were dependent upon a daily supply of fresh food materials.

The art of preserving foods depends upon the science of bacteriology. A more complete knowledge of the causes of decomposition and methods by which they may be prevented has enabled us to perfect the crude and primitive methods that have been in use from time im-

memorial, so that it is now possible to preserve certain foods practically indefinitely without in any way injuring their nutritive value or seriously interfering with their appearance and taste.

The chief harm has come from the blind use of chemical germicides, without regard for their harmful properties. The simplest and cheapest way to preserve food is by adding one of these chemicals, and the method was, therefore, seized upon by alert men whose chief interest was of the pecuniary kind. The question was to find the smallest percentage of a chemical which would prevent the decay of some particular food product, trusting to luck that the preservative used might prove harmless to the consumer. Often these chemicals were added with a liberal hand; further, it was soon found that chemical preservatives could be used to preserve food products for the market from materials already so decayed as to be unsalable in their original condition.

The National Pure Food and Drugs Act of 1906 was passed largely to meet this situation. This law considers any food which contains some "added poisons or other deleterious ingredient which may render such article injurious to health" as adulterated. To Harvey W. Wiley belongs the credit of inducing Congress to pass this legislation against opposition and for an aggressive administration that proved useful in bringing the whole question prominently before the public.

Cold.—Cold must be regarded by the sanitarian as an antiseptic rather than a germicide. Low temperatures kill few bacteria, but prevent the growth and multiplication of most of them. Even the antiseptic properties of cold are not as marked as they were once believed to have been.

Some bacteria grow and multiply at low temperatures, even at 0° C. In 1871 Burdon-Sanderson was the first to show that freezing does not kill bacteria. Von Frisch demonstrated that subjecting a putrefying solution to a temperature of —87° C. for some hours did not effect sterilization. Leidy in 1848 showed that water derived from melted ice contained not only living infusoria, but also rotifers and worms. Pictet and Young found that anthrax and symptomatic anthrax cultures were not killed after an exposure of 108 hours to —70° C. Later MacFayden proved that the temperature of liquid air does not kill bacteria; he subjected cultures to temperatures of —315° F. Ehrlich has recently shown that cancer cells kept cold will live and remain virulent for at least two years.

While no microorganism pathogenic for man will grow and multiply at the low temperatures of the refrigerator, there are a number of saprophytic bacteria and molds that develop abundantly at temperatures as low as 0° C. Milk, meat, eggs, and other products kept in cold storage at or near the freezing point may show a notable increase

in the number of bacteria. A number of tests made in my laboratory showed that in the case of milk these low-temperature microorganisms belong mainly to the putrefying and proteolytic group. They produce an alkaline reaction in the milk and a bitter taste. Whether they are capable of forming poisonous products at these low temperatures is doubtful.

For the most part pathogenic bacteria withstand freezing temperatures. They, however, suffer a quantitative reduction when frozen (see Ice and the effects of freezing upon bacteria, page 887). Most animal parasites die in cold storage; a few, however, survive. The time in which the material has been refrigerated is an important factor. Just as water becomes safer by storing it, so with foods, but cold storage foods, while safer, cannot be entirely relied upon. Thus, cold appears not to affect trichina. *Tænia saginata,* the beef tapeworm, dies in twenty-one days, and *Tænia solium,* the pork tapeworm, may live more than twenty-nine days in cold storage.

Fortunately, cold causes a quantitative reduction in the number of harmful bacteria, even though it does not produce complete sterilization. The element of time here plays an important rôle, as most of the surviving pathogenic microorganisms soon die. From a sanitary standpoint the protection afforded by refrigeration is reassuring, although not perfect.

The best temperature at which foodstuffs may be kept must be determined for each case. Some substances, such as meat and poultry, are better preserved when actually frozen; others, such as shell-eggs or milk, are materially injured by freezing. In any event, the temperature of the icebox should not rise above 7° C. At this temperature bacterial growth does not entirely cease, although very markedly hindered. Few household refrigerators reach this temperature or maintain it for any length of time—either through faulty construction or on account of insufficient ice. Often the icebox is placed in a sunny corner, or, for convenience, near the kitchen stove. The doors of the ice chest frequently do not fit well, which results in needless waste and imperfect refrigeration. A study of household refrigerators discloses the fact that the temperature is often 15° C. and higher. Such conditions make good incubators, favoring bacterial growth. The necessity for scrupulous cleanliness, aeration, and dryness in all refrigerating devices needs only be mentioned.

In ordinary refrigerating plants moisture condenses on the surface of the objects exposed. In the case of meat this moisture dissolves some of the proteins, extractives, and salts, and makes a perfect culture medium for bacteria and molds. In the case of meats it is, therefore, better to hang them in a current of dry, clean air, in order to desiccate the surface, before they are placed in the refrigerator. The

dried surface delays the inward growth of the inevitable bacterial contamination upon the surface.

Articles of food may be kept in a satisfactory condition in cold storage for a very long time. The time varies with the article and its condition when placed in storage, also with the temperature and other factors. A striking illustration of the great preserving power of low temperatures occurred several years ago in Northern Siberia. In consequence of a great landslide on the banks of the Kolyma, the head of a mammoth became exposed and was so well preserved that even the fleshy trunk remained. It is said that famished wolves and half-starved natives began to eat of the flesh. The Russian government sent Dr. Hertz to rescue what remained. The mammoth had remained in cold storage perhaps thousands of years. Some of the soft parts are now preserved in the Museum at St. Petersburg. This must not be taken as justification of prolonged storage or the "cornering" of foods for economic gain in mammoth cold storage warehouses. While meat, poultry, eggs, and vegetables may be kept in a satisfactory condition for months and transported over seas, cold storage need not be unduly prolonged. In any case, the consumer is entitled to know whether the article is fresh or stored, and the time it has been in cold storage. These facts should be stated upon the label or stamp.

During the past few years cases of so-called "ptomain" poisoning have been attributed to the ingestion of cold storage poultry. It is supposed that the undrawn condition stimulates decomposition during cold storage. Laws have, therefore, been passed in certain states requiring poultry to be drawn before being placed in cold storage. It is claimed, on the other hand, that the undrawn fowl keep better—it is certain that they weigh more, which is an advantage to the dealer. The disadvantage of drawing fowl by the ordinary method is that the carcass becomes contaminated with the intestinal contents and putrefactive processes are hastened. This question was investigated by the Massachusetts State Board of Health, and the conclusion was reached that it made practically no difference whether the fowl were drawn or not, but that they must be perfectly fresh when placed in cold storage. Poultry is kept below 0° C., at which temperature no noticeable change occurs. It was found that cold storage fowl are even less contaminated with bacteria than freshly killed birds that have hung for a few days. However, the cold storage animals, when removed from the refrigerator, decompose more quickly than the fresh.

Contrary to what might be expected, drawn poultry decomposes more rapidly after removal from cold storage than undrawn. This is because in the process of drawing the intestines are broken below the gizzard and the carcass becomes badly contaminated with intestinal bacteria. If the entire alimentary canal, esophagus, crop, gizzard, and

intestines are removed intact, and with due care to prevent bacterial contamination, the bird is practically safe from putrefaction. In case, therefore, poultry is drawn before it is placed in cold storage, the drawing should be done with bacteriological care.

From a sanitary standpoint, then, refrigeration is one of the best methods of preserving foodstuffs. The advantages of cold as a preservative are that it neither adds any constituent to the food nor takes away any constituent from the food. Cold imparts no new taste, nor does it seriously alter the natural flavor. It does not diminish its digestibility nor cause a loss of nutritive value. The material is left in approximately its original condition. Cold may, therefore, be regarded as one of the simplest and best antiseptics we have for the preservation of foods. It is now almost universally applied to prevent decomposition and decay. The housewife uses it to keep food in cold cellars, deep wells, and the like. During the last fifty years the use of ice for the purpose of refrigeration has become commonplace. Fresh and wholesome food may now be transported to the tropics, and the sustenance of large communities in insular and arid regions is made possible and pleasurable through the preserving use of cold.

Drying.—Drying, desiccation, or evaporation is a favorite and primitive method of preserving meats, fruits, vegetables, and various food materials. Dryness furnishes ideal antiseptic conditions. Microorganisms must have moisture to grow and multiply. Most pathogenic microorganisms soon die when dried, hence the process has a decided sanitary advantage. Further, dried fruits, vegetables or meats are rarely eaten raw, and the cooking is a further sanitary safeguard.

The effectiveness of drying as a food preservative depends upon the thoroughness with which the process is carried out. It is not so well adapted to meats as to vegetables and fruits. Dried meats lose their natural flavor, which may be replaced with others less real. All sorts of organic foodstuffs, even the most decomposable, such as milk, eggs, or meat, may be dried and, if kept dry, they will keep in a satisfactory state almost without limit of time.

Theoretically dryness is not a complete safeguard, for the reason that a few microorganisms survive, particularly bacterial spores. Despite this slight limitation, it is more than reasonably safe and an entirely satisfactory procedure. Practically the only change in dried foods is the loss of moisture, which may readily again be supplied. Dryness has the great advantage in that no added chemical or added preservative process is necessary; further, dried foods are quite as nutritious and usually quite as digestible as the fresh articles, although not quite as savory.

Dried Meat.—In the dry climates of South America and on our western plains meat is cut into thin strips, suspended in the air,

and exposed to direct sunlight. In a short time the moisture disappears and the hard dry pieces keep indefinitely, or as long as they are kept dry. The meat retains a fair degree of palatability and practically all of its nutrient properties. This is known as jerked beef.

Dried beef is also prepared by first treating the meat with condiments and then drying it artificially. Chipped beef or dried beef is prepared in this manner, except that the meats are often smoked as well as salted and desiccated, so that in their method of preparation more than one method of preservation is employed.

Powdered meats are prepared by complete desiccation, and such products are found upon the market as a finely ground powder. Meat powders are made not alone from fresh meats in their natural state, but are also prepared after more or less artificial digestion.

Dried Fruits.—Dried apples are taken as a type of dried fruits and vegetables. The apples may be dried naturally by cutting them into convenient sizes and exposing them to the action of the sun. This is more a domestic than a commercial industry. When apples are dried by this simple process they darken and become unattractive in appearance. This is due to the oxidizing action of the enzymes when exposed to the air. When properly prepared the dried apple has its moisture content reduced to approximately 30 per cent. or less.

In order to prevent the darkening of the surface during the long exposure necessary to secure the proper degree of evaporation, apples are usually subjected to the fumes of burning sulphur. The sulphur dioxid acts as a bleaching agent and the sulphurous and sulphuric acids retained in the apple act as preservatives. Apples treated with sulphur fumes are less likely to decay or become infected with molds than a similar product not exposed to sulphur fumes. The process is objected to from the standpoint of health, for the reason that the sulphurous acids and sulphites are admittedly injurious to health. The Department of Agriculture found that approximately half of the evaporated fruits purchased on the open market had been treated with sulphur fumes. In order to obtain a satisfactory dried product it is of some importance that the fruits be selected, so as to reject all imperfect, rotten, or infected specimens.

Evaporated apples is a term applied to apples dried artificially instead of being exposed to the sun's heat. The process is rapid and satisfactory, and has no sanitary objections.

Dried Eggs.—Eggs are broken out, mixed and dried by spreading the mass in a thin film in shallow pans or upon a broad revolving belt; the water is abstracted by exposure to a current of warm dry air. The egg substance may also be dried by forcing it through small orifices under a high pressure into a drying chamber so adjusted as to temperature and size as to secure the desiccation of the minute particles of egg

spray before they fall to the bottom. Egg substance thoroughly dried keeps satisfactory in almost any climate. It retains all the nutritive value in the original egg.

Dried Milk.—Milk must be dried quickly and at a comparatively low temperature in order to obtain a successful product. It must be dried quickly in order that it will not spoil during the process, and the temperature must not be high enough to coagulate the lactalbumin, otherwise the addition of water would not restore the milk to its former homogeneous state. Milk is sometimes dried in a very thin film on metal plates; sometimes *in vacuo*. In this way the milk can be reduced to a dry state in a very short time and without reaching a temperature sufficiently high to produce physical changes. Another method of drying milk consists in atomizing it under pressure and projecting it into a warm chamber, the temperature of which is so regulated that the fine particles are completely deprived of their water before they reach the bottom of the vessel. The milk is thus reduced almost at once to a fine powder. Dried milk when mixed with water is practically restored to its original condition. Milk powder should be either kept in a cool place or sealed in air-tight packages in order to prevent the fat becoming rancid. Dry powdered milk properly cared for will keep almost indefinitely. Since practically 88 per cent. of milk is water, there is a decided economic gain, so far as the handling and transportation are concerned. Powdered milk should, of course, be made from milk derived from healthy cows handled under sanitary conditions and free from infection. The milk may be pasteurized before it is reduced to a powder. Powdered milk is finding an increasing and legitimate field of usefulness for cooking, household purposes, and even as a beverage for adults. It should, however, not be depended upon for infant feeding.

Salting and Pickling.—The preservation of meat with brine or common salt is one of the oldest processes known. The brine should contain from 18 to 25 per cent. of salt. For red meats a little potassium nitrate is often added; this salt has slight antiseptic properties, but brings out the red color. In the processes of salting some of the meat protein, bases, and extractives are dissolved out and the fibers become hardened; the nutritive value and digestibility, therefore, is somewhat diminished.

Pickling includes preservation of food in brine, vinegar, weak acids, and the like. These substances have antiseptic and also feeble germicidal properties, depending upon their concentration.

Pickled meats are prepared by soaking meat, especially pork, in a brine made of common salt, though other substances, such as sugar, vinegar, and spices, are often added. Chemical preservatives are sometimes added to the brine. Those most frequently used are sulphite of

soda or boric acid. With proper methods these added chemical antiseptics are not necessary. The vinegar which is employed, or acetic acid, may be injected into the carcass before it is cut up. When the arteries are filled with vinegar in this way it rapidly permeates to all parts of the meat and acts as an excellent and unobjectionable preservative in cases where an acid taste is desired. It is stated that carcasses which have been injected with vinegar are easily preserved and require far less salt and other condimental substances than when not so treated. The process has no sanitary objections.

Trichina die after a prolonged period of pickling. Cysticerci are killed in 21 days' exposure to 25 per cent. brine. Many pathogenic bacteria die in brine of this concentration, but the salt must be looked upon as an antiseptic rather than a germicide; that is, it prevents growth rather than kills the bacteria that are present. From a sanitary standpoint there is some, though slight, danger of conveying infection in foods that have been improperly salted or pickled. Attention is called to the fact that the cases of botulism studied by von Ermengen were caused by a ham kept in brine under conditions favoring anaerobic growth.

Decomposition may also be arrested by the use of syrups, which have an entirely similar action to that of salt, vinegar, or weak acids; that is, a strong solution of sugar will prevent growth, but cannot be depended upon to kill parasites. However, most of them die under such conditions in the course of time. As most preserved foodstuffs are cooked before eaten, there is small danger in articles prepared by these processes.

Jellies and Preserves.—By preserving is commonly understood the addition of a large amount of sugar. The principal preserves are jellies, marmalades, jams, and fruit butters. These substances are entirely free from the danger of conveying infection, not only on account of the antiseptic action of the sugar, but for the further reason that they are always cooked in preparation. Jellies are frequently adulterated by the substitution of apple stock. Apples contain a large number of pentose bodies which favor jellification. A common method of manufacturing jelly for the trade has been to use a stock of apple juice or cider, or a preparation made from the cores, skins, and rejected portions of the apple at evaporating factories, or from whole rejected apples. This stock is used as a common base for the manufacture of jellies of different kinds. Apple juice used as a substitute for other fruit juices in the making of preserves is a common fraud and an adulteration, according to the Food and Drugs Act, unless plainly stated upon the label. Phosphoric acids and other acids are added to jellies to enable jellification to take place with the use of less fruit and more water. Jellies are also adulterated with artificial color-

ing matter, particularly the coal-tar dyes. Artificial flavors which closely resemble the particular flavor desired are sometimes employed. The chemical preservatives most frequently added to jellies and preserved fruits are salicylic acid, benzoic acid, or benzoate of soda.

Smoking.—The smoking of fish, beef, hams, and other food products consists mainly in rapid drying plus the germicidal action of certain substances in the smoke.[1] The meat or fish is exposed to the smoke of a smoldering wood fire of oak, maple, or hickory, usually after a preliminary salting. The articles so exposed become dry and impregnated with pyroligneous products—acetic acid and creasote, formaldehyde, and other germicidal substances. The penetration is only partial.

An artificial or quick method of smoking meat is to brush the pieces or dip them in pyrolignic acid at definite intervals, and finally dry in the air. The effects of the smoking do not penetrate very far; therefore, in sausages of generous diameter putrefaction often occurs in the interior. Smoked sausage may, therefore, be dangerous, as far as various parasites and the products of decomposition are concerned, and the same is true of smoked ham and other meats exposed in large pieces. As smoked meats are often eaten raw, the occasional survival of parasites in such products has some sanitary significance.

Canning.—The process of canning is practically synonymous with sterilization and is, therefore, one of the best sanitary safeguards we have against parasites and the injurious products of putrefaction in foodstuffs. The process of canning was discovered by M. Appert of Paris in 1804, long before the days of bacteriology. Appert found that meats and other foods in sealed vessels would usually keep indefinitely if, after being sealed, they were kept for an hour in boiling water. He improved the process in 1810 by introducing a method of sealing the cans after the heating process had driven out the air and replaced it with steam, so that when cool a vacuum is formed. For all practical purposes this is the universal method of canning to-day, except that now the cans are given a second heating, after an interval of a day, in order to permit the germination of spores and the destruction of all spore-bearing bacteria. In other words, canning is a practical application of the well-known laboratory method of fractional sterilization.

The objection is sometimes raised that the contents of the can are improperly sterilized and that the surviving spores germinate at the first opportunity and cause decomposition. Fortunately, an improperly sterilized can of food tells its own story, and the gaseous products of putrefaction may even burst the tin or leave the food in such condition that when the can is opened it would be so offensive to the sense of

[1] The process was probably accidentally discovered in connection with crude attempts to use artificial heat for drying purposes.

smell that no one would use it. The process of canning fortunately does away with the necessity of using chemical preservatives of any kind. The proper authorities should be authorized to prohibit the canning of foodstuffs that have already undergone perceptible decomposition, or, if not injurious to health, they should be labeled "second quality." The law should require that the quantity contained within the can and the date on which it was put up as well as the amount should be stamped in the tin. This phase of the question is perhaps more of economic than of hygienic importance, but will be required in time as surely as the present law now requires honest labeling in other particulars.

Sometimes scraps or inedible portions of diseased or decayed meats are canned and the flavor disguised. This cannot be too severely condemned if sold as first quality.

Before meats are canned they are first parboiled for eight to twenty minutes, in order to secure the shrinkage before the meat is placed in the can. In the parboiling there is a certain loss of fat, soluble mineral matter, meat bases, and water. However, the shrinking of the meat concentrates it, as far as nutritive value is concerned, and, therefore, compensates for the loss. The parboiled meat is then placed in the tin and a small quantity of the soup liquor added. The cans are closed and soldered and then placed in autoclaves and subjected to steam under pressure. Usually a small hole is left in the can in order to permit the exit of air and gases. This is sealed off at once after heating. The cans are then subjected to a second heating at 225° to 250° F. for one to two hours. A modified process consists in placing the cans upon an endless conveyor which exposes the can to a high temperature in an oil bath a sufficient length of time to sterilize the contents at one exposure.

In Germany tuberculous and trichinous meat is sterilized and sold as second quality meat in accordance with the third class or "*freibank*" meat system. There is no known sanitary objection to this practice, provided the sterilization is complete and the label represents the true nature of the product.

Canned foods are sterile foods and, therefore, generally safer than fresh foods. Fresh foods, of course, are to be preferred to those that have been sterilized, although many unsterilized foods are more dangerous in the fresh state than after they have been exposed to a high temperature. The process of canning, discovered by Appert and afterward perfected through the work of Pasteur, has proven of inestimable benefit to mankind. It enables nourishing food of a perishable character to be kept and transported to great distances and to be used in localities where fresh foods are unobtainable. Without this method of preserving foods the pioneer and the explorer would be seriously

handicapped. Large army and navy maneuvers would be materially impeded, and great metropolitan cities would be impossible. Wiley states that "the winning of the West has been marked by the débris of the rusty can."

Canned foods are not only safe, but are quite as nutritious as the original articles. The process permits us to have a well-balanced ration throughout the year—irrespective of season. The canning industry is growing to enormous proportions, and, on account of the great importance of the process, the character and quality of foods thus preserved should be wholly above suspicion, and no adulteration or sophistication of any kind permitted. Every can should be plainly stamped with the quantity and true nature of its contents and also the date when it was first sterilized.

Concerning the character of the container Wiley states: "Much in the direction of securing a better product may be accomplished by a more careful selection of the container. The common method of preserving canned goods is in tin. This material, as well known, is placed on the surface of sheet iron and should be free of other metals. Lead especially should be excluded from the composition of the tin as far as possible. In spite of all these precautions, however, the coating of the tin is sometimes broken, so that the iron itself may be attacked, perforations result, and the package of goods be spoiled. More frequently, however, the erosion of the tin plate occurs over widely extended areas, introducing into the contents of the package a considerable quantity of tin salts. This may be prevented to a certain degree by coating the surface of the tin with a gum or varnish which is not acted upon by the contents of the package. Glass is also coming into more general use, and if it could be secured of a character to avoid breakage it would be possible to replace to a considerable extent the tin packages now in such common use, and thus prevent the introduction of soluble tin salts into food. In this case the glass itself should be free of lead, borax, and fluorids. A glass package is now coming into use which is tough and resistant to ordinary causes of fracture. Much may be expected from progress in this direction."

Chemical Preservatives.—Chemical preservatives are nothing more nor less than antiseptic substances; that is, substances which restrain the growth and development of bacteria and molds. Chemical preservatives in the proportions commonly used may have little or no germicidal action. Such substances as sugar, salt, vinegar, vinegar extract of spices, and the pyroligneous products in wood-smoke are not regarded as "chemical" preservatives, but as "natural" preservatives or condimental substances, although their mode of action is precisely the same as the chemical preservatives. There is a great prejudice against the use of any preservative for our foods if this preservative is a "chemical" or

"drug," whereas no objection is raised to the same substance if derived from "natural" sources. Thus, foods exposed to a smoldering wood-fire become impregnated with pyroligneous acid, which includes creasote, acetic acid, and probably formaldehyde and other substances having antiseptic properties. This method of food preservation is not only countenanced by the law, but is favored on account of the savory result and the antiquity of the process.

The great increase in the use of chemical preservatives in foods during the last fifty years is owing to the fact that this is the cheapest and surest method of preservation, thus offering a convenient method of supplying the needs of large communities as well as remote places. The question, therefore, has an economic side that cannot be disregarded. Here, however, we must confine ourselves to the health aspect of the problem. Fortunately we possess two efficient and wholly unobjectionable processes for the preservation of food, viz., refrigeration and sterilization by heat, which for the most part make it unnecessary to resort to the use of chemical preservatives. One of the most objectionable uses that can be made of chemical preservatives or any other method of food preservation is to conserve foods which are so decayed as to be unfit or possibly injurious to health if used fresh. The law cannot be too strictly enforced in order to prohibit the use of chemical preservatives and condiments used to disguise such foods, which may then be sold at high prices as first quality.

Upon general principles it is undesirable to add a chemical substance of whatever nature to food for the purpose of preserving, coloring, or improving its appearance, and in most countries this practice is prohibited by law. There are, however, a few instances in which the addition of some chemical preservative in minimal amounts seems harmless, and occasionally even desirable, as, for example, small quantities of benzoate of soda in catsup; a thin film of gum benzoin as a protective coating for chocolate, etc.

No sweeping generalization can be made concerning all chemical preservatives. Each substance must be considered for itself, and each substance must further be considered in relation to the particular foodstuff for which it is proposed. It may, however, be stated as a general rule that any chemical which is poisonous in large amounts should be considered as poisonous in small amounts until the contrary is proven. In other words, the consumer is entitled to the benefit of the doubt. The toxicology of various food preservatives is in its infancy and frequently presents a very difficult and complex problem. Thus, lead in one large dose is not particularly harmful. The older practitioners frequently gave twenty, thirty, and more grains of sugar of lead (acetate of lead) for diarrheal affections. Only a minute portion of the lead taken in one large dose is absorbed; the rest is quickly eliminated.

However, if the same amount of lead should be taken in small subdivided daily doses, it would be absorbed, retained by the tissues, and the poisonous action would be cumulative, so that serious chronic lead intoxication would result. On the other hand, hydrocyanic acid, one of the most poisonous chemicals known, is harmless in small amounts, for the reason that when introduced into the body it meets the available sulphur (H_2S), with which it unites to form a sulphocyanid, as KSCN. The potassium sulphocyanid is not poisonous, and it has been shown experimentally that animals are able to withstand larger quantities of hydrocyanic acid by first giving them substances which increase the available amount of sulphur to form this chemical combination. Benzoic acid in large amounts is irritating and produces well-defined symptoms of poisoning; small amounts of benzoic acid are paired in the liver and eliminated by the kidneys as hippuric acid, a normal and harmless constituent of the urine.

The point at issue now is to determine which of the chemical substances are injurious to health. In the present transitional state of our knowledge it is not possible to make a final statement concerning all or perhaps any one of them. It is well known that the most serious poisons may be taken in minute amounts without apparent injury. In fact, many medicinal substances in the pharmacopeia are very poisonous, but in therapeutic doses may be quite beneficial. The effect of the continued use of chemical substances in small amounts will require long and patient observation to determine whether or not they should be permitted as food preservatives. Of all the substances so far brought forward, the least harmful is benzoic acid and benzoate of soda. There can, however, be no defense for the use of formaldehyde, salicylic acid, sulphites, and a host of other chemicals. So far as we know the human organism possesses no natural mechanism for rendering them harmless.

There can be no defense for the use of chemical preservatives to hide inferiority. This is well illustrated in the case of bleached flour. The only purpose of the bleaching is to make the flour from a dark wheat look as white as the best patent flour. It was recently discovered that this "artificial aging" of flour may be accomplished by adding nitrogen peroxid. The flour absorbs this poisonous gas as a sponge absorbs water and instantly becomes white. Processes of this kind should be regarded as a common fraud, for the flour is not improved in any way except in appearance, which is, after all, a deception. The silly process of modifying the natural colors of food is illustrated in the use of copper sulphate to give peas a bright green hue, and the use of anilin dyes in glucose, jellies, fruit juices, ices, and other substances to imitate the color of natural flavoring extracts. "Natural" colors, such as caramel and vegetable substances, are also frequently used. The substitution of cheap chemicals for high-priced natural flavoring extracts, the substitu-

tion of acetic acid or even mineral acids for genuine vinegar, the substitution of saccharin for sugar, the paraffin polishing of rice, and similar devices are nothing but common frauds, which may in some cases also be injurious to health.

Benzoic Acid and Benzoate of Soda.—Benzoic acid is an organic acid contained largely (12 to 20 per cent.) in gum benzoin, and also in balsam of Peru and balsam of Tolu. It is obtained from gum benzoin, from the urine of herbivorous animals, and artificially from toluen, by treating it with chlorin and heating with water to 150° C.

The storm center of the question of chemical preservatives in this country has raged about the use of sodium benzoate. Wiley conducted experiments upon a number of healthy individuals known as the "poison squad." These men were given rather large quantities of sodium benzoate with their meals and the result seemed to be an impairment of the appetite, disturbance of digestion, and other injurious effects in certain instances. On the other hand, the Referee Board appointed by President Roosevelt and consisting of Remsen, Chittenden, Long, Taylor, and Herter found that moderate quantities over a period of four months have no appreciable influence upon health.

The reason why benzoic acid in moderate amounts is believed to be harmless is that the body possesses a special mechanism for taking care of this substance. Many of our ordinary foods contain substances which are transformed in the body into benzoic acid. Some foods, such as cranberries, contain this acid in notable amounts. Benzoic acid meets glycocoll (one of the decomposition products of protein) in the liver. Benzoic acid and glycocoll form hippuric acid, a normal and harmless constituent of the urine. We, therefore, know that the human organism is prepared to take care of and render harmless a certain amount of benzoic acid; we know that this mechanism is a very efficient one, and is capable of taking care of relatively large amounts of benzoic acid.

There can be no serious objection from the standpoint of health to the addition of 0.1 per cent. of sodium benzoate to catsup, on account of the small quantity of this article consumed at any one time, and further on account of the long time a bottle of catsup is usually kept after it is opened in the household. There is, thus, the added economic gain of preserving the catsup until it is all consumed. The same object may be obtained by the use of a sufficiently strong vinegar extract of spices, but the question may be asked whether the aromatic and preserving substances in the vinegar extract of spices may not be more irritating than the sodium benzoate.

Hoffman and Evans[1] have shown that ginger, black pepper, and cayenne pepper fail to prevent the growth of microorganisms. Nutmeg and allspice have slight antiseptic properties, but only for a very few

[1] *Journal of Industrial and Engineering Chemistry*, Nov., 1911, p. 835.

days. Cinnamon, cloves, and mustard, on the other hand, have very marked antiseptic powers and are valuable preservatives. The active antiseptic constituents of mustard, cinnamon, and cloves are the aromatic or essential oils which they contain.

No one would advocate the promiscuous use of sodium benzoate in foodstuffs generally. Its use in such foods as cider or tomato soup may be questioned on account of the amounts that would be taken in such articles. Further, benzoate of soda placed in an acid medium becomes benzoic acid. It is difficult to know where to draw the line, and the consumer must be given the benefit of the doubt, but the evidence seems fairly well established that in the case of benzoate of soda small amounts are harmless.

The question has a large economic significance in addition to its sanitary aspect, for it is claimed that benzoates as well as other chemical preservatives permit the use of rotten tomatoes, skins, and undesirable food which otherwise could not readily be preserved. Benzoate of soda is a rather feeble germicide at best, and in such dilute proportions as 0.1 per cent. has little antiseptic power.

Borax and Boric Acid.—Both boric acid and borax are only mild antiseptics. They are not very potent germicides. They are generally used together, for the reason that the combination of the two is more efficient than either one alone. Locally boric acid is not very irritating, and for this reason it has been extensively used in surgical practice. To some skins, however, it is very irritating, and cases are reported of its absorption from wounds and cavities when used too freely, causing depression and eruptions, such as erythema and urticaria. Fatal results have been reported in a few cases from injecting the solution into abscess sacs, and from washing out the stomach with it.

Boric acid and borax are used for preserving meats, milk, butter, oysters, clams, fish, sausage, and other foods. For meat it is often mixed with salicylic acid and applied externally. For milk it was a common practice to add to one quart of milk 10 grains of a mixture of equal parts of borax and boric acid; for butter the amount used is about one-tenth of an ounce to the pound.

The effect of small amounts of boric acid and borax upon healthy human beings has been extensively studied and has resulted in conflicting testimony.

On one hand we have the researches of Chittenden [1] and Liebreich [2] with dogs fed upon articles containing borax and boric acid. To say the least, in both series the digestion of the food was not notably impaired and the animals gained in weight. The same result followed the experiment made by Liebreich upon rabbits and guinea pigs. No

[1] *American Jour. of Physiology*, 1898.
[2] *Vierteljahresschrift für gericht. Med.*, 1909, also *Lancet*, Jan. 6, 1900.

injury appears to have followed the administration of boric acid to pigs, calves, and children by the British Commission.[1] Tunnicliffe[2] made experiments from which he inferred that neither borax nor boric acid affected the health of the children experimented on. Vaughan and Veenboer[3] conclude that in the small amounts required for preserving cream and butter, and that used as an external dust on hams and bacon, both boric acid and borax are unobjectionable from a sanitary standpoint.

On the other hand, the experiments made by H. E. Annette[4] led him to an opposite conclusion. He found boric acid injurious to kittens, and naturally assumed that the use of milk containing it might be hurtful to young infants. Foster and Schlenker[5] found that albumin digestion was impaired by boric acid, which also produced increased desquamation of the intestinal epithelium. Doane and Price[6] made experiments on calves which indicate that borax and boric acid in milk retard digestion to a slight extent.

As these substances are not normal constituents of the body, nor are they normal constituents of foods, the conservative course would be to avoid their use until satisfactory evidence has been adduced that they are free from harm in the amounts commonly used for preserving food.

Formaldehyde.—Formaldehyde has been and still is used extensively as a preservative for milk and other articles of food. Formaldehyde in large quantities is exceedingly irritating, and death in isolated instances has been reported from the swallowing of amounts from 1 to 3 ounces. There has been much discussion as to the effect of the small quantities ordinarily used as a food preservative. Bliss and Novy[7] and Halliborton[8] have shown conclusively that small quantities of formaldehyde greatly delay the digestion of proteins by the gastric and pancreatic juices, the digestion of starch by the pancreatic juice, and the curdling of milk by rennet. It is also known that some individuals are especially susceptible to the effect of formalin, small quantities in the food causing dyspepsia and other disturbances of digestion. Formaldehyde unites directly with protein matter to form new compounds of an undetermined nature. Thus, formaldehyde added to egg albumin prevents its coagulation by heat, and added to gelatin prevents liquefaction. It hardens tissues, so that it will render fish and meat tough and brittle, even in proportions as dilute as 1-5,000, hence

[1] *Vierteljahresschrift für gericht. Med.*, 1901.
[2] *Journal of Hygiene*, 1901.
[3] *American Medicine*, March 13, 1902.
[4] *Lancet*, Nov. 11, 1899.
[5] Quoted in report of Kober on "Milk Preservatives," U. S. Senate Commission, 1902.
[6] *Bulletin No. 86*, Maryland Agricultural Experiment Station, Sept., 1902.
[7] *Jour. of Exp. Medicine*, 1899, Vol. IV, p. 47.
[8] *British Medical Jour.*, 1900, Vol. II, p. 1.

it is not generally applicable as a food preservative. In small amounts it delays decomposition; in large amounts it is an active germicide. Its use in milk was recently advocated by no less an authority than von Behring, but this view met with almost unanimous protest.

There can be only one opinion concerning the use of formaldehyde in foods, and that is absolute condemnation of the practice. It is prohibited by the statutes of practically all nations having pure food laws.

Salicylic Acid.—Individuals differ greatly in their susceptibility to salicylic acid. In mild cases of poisoning with this substance there is a feeling of fulness in the head with roaring sounds in the ears, dimness of vision, profuse perspiration, confusion, and dulness. Large doses of the acid cause intense irritation of the throat and stomach, leading to vomiting and difficulty in swallowing. Later there may be diarrhea. Eczema and other skin eruptions may appear, and dimness of vision and deafness may continue for some time. The long-continued use of salicylic acid and its salts has led to a form of chronic poisoning in which the chief symptoms have been loss of appetite, diarrhea alternating with constipation, irritation of the kidneys, skin eruptions, and mental depression. Such results are said to have followed the use of articles of diet preserved with salicylic acid. The use of such foods may be objectionable in the case of aged, feeble, and susceptible persons. Salicylic acid and the salicylates are more efficient antiseptics than boric acid or borax, but they are not used extensively on account of the taste, or rather the tendency to cause unpleasant flavors. They are for the most part used in jams, fruit juices, soda water syrups, cider, wines, and other sweet preparations. The objection to the use of salicylic acid in food is practically unanimous and well founded.

Sodium Nitrate.—Sodium nitrate or potassium nitrate (saltpeter) is not used as a preservative, but as an indirect coloring matter. It retains and accentuates the red color of meat. It is not known to be harmful in the small quantities in which it is commonly employed, but must be regarded as a fraud when used to make stale meat look fresh.

Potassium Permanganate is also used on the surface of meat to destroy decomposition. This may be detected by heating a knife in hot water, plunging it into the meat, and withdrawing it quickly. This brings out the hidden odors of putrefactive changes.

Sodium Fluorid.—Sodium fluorid has considerable antiseptic power, putrefaction being delayed by the addition of 1 part to 500; and 1 in 200 arrests completely the growth of bacteria. The fluorids are absorbed from the alimentary canal and are excreted by the urine, but this takes place very slowly, and much of the fluorid is stored up in the body, some in the liver and skin, but most in the bones in the form of calcium fluorid. Crystals of this very insoluble salt are found

in masses in the Haversian canals, which increases the hardness and brittleness of the bones.

Sulphites.—Sulphites act as antiseptics and also preserve the red color of meats. Sodium sulphite and bisulphite and sulphurous acid are used principally upon fresh meats, where they act as a preservative and as a retainer of color. Sulphur dioxid is also much employed for the bleaching of fruits. Sulphites, even in minute amounts, interfere with the action of ferments, and thus influence digestion. Free sulphurous acid is very irritating. Sodium sulphite is very poisonous when injected subcutaneously or intravenously. Death occurs by paralysis of respiration. Much larger quantities are tolerated by the mouth, the sulphite being slowly absorbed. The greater part is converted to the harmless sulphate during and after absorption. The quantities ordinarily used in preserved food cause no immediate symptoms, even when continued for several months. If, however, the animals are killed and examined, extensive hemorrhagic and inflammatory lesions are found in various organs.[1] These lesions are probably due to destruction of red blood cells or infarction. Harrington in 1904 also described nephritic changes. In 1898 the Imperial Board of Health in Germany forbade the use of sodium sulphite in food on account of its dangerous properties, and it is also forbidden by our Federal Pure Food Act of 1906.

Sodium Bicarbonate.—Sodium bicarbonate is too ineffective as a germicide for general use as a food preservative. It is sometimes added to milk in order to neutralize the excess of acid.

Hydrogen Peroxid.—Hydrogen peroxid is perhaps one of the less dangerous of the chemical preservatives, and is considered by some to exert no deleterious effect whatever in the quantities commonly used. It is used for the preservation of wine, beer, and fruit juices, and also in milk.

Arsenic.—Arsenic in food comes from a variety of sources. Glucose is apt to contain it, especially if impure acid is used to hydrolize starch in the production of glucose. This was the source of the arsenic in the beer which caused the epidemic of peripheral neuritis several years ago in England. Arsenic may also contaminate certain anilin dyes as well as shellac,[2] which is now so much used as a coating for some kinds of cheap confectionery and bakers' goods, and also as a varnish on receptacles and containers of various kinds.

The use of preservatives containing lead, arsenic, or other substances known to be poisonous finds no advocates.

[1] Kionka and Ebstein, 1902.

[2] Smith, B. H.: "The Arsenic Content of Shellac and the Contamination of Foods from This Source," *Cir. 91*, U. S. Dept. Agr., Bureau of Chemistry, Washington, 1912.

THE PREPARATION OF FOOD

Cooking.—Cooking may be regarded as the greatest sanitary innovation ever introduced by man to protect himself against infection. The heat required for thorough cooking kills all forms of infection and, therefore, renders food safe, so far as these dangers are concerned. The heat also destroys most of the toxic products of decay; thus, the true bacterial toxines are destroyed at temperatures of about 60° C. Foods may sometimes contain heat-resisting poisons. Thus, boiling has no effect upon muscarin, the poisonous principle in certain toadstools. Heat also does not destroy a poisonous principle sometimes found in mussels. The colon bacillus and other microorganisms produce thermostable substances that are poisonous when injected into the lower animals, but the relation of these heat-resisting toxic substances to food poisoning in man is not at all understood. It is highly improbable that foods ordinarily contain heat-resisting poisons resulting from bacterial decomposition.

Trichina die at 65° C.; cysticerci, or the larval stage of tapeworms, at 52° C.; the non-sporulating bacteria are for the most part destroyed at 60° C. Food thoroughly cooked throughout will always reach these temperatures, but much meat and many vegetable food substances are preferred rare or underdone, and, while the outside of a large piece of meat may be thoroughly cooked or even charred, the interior may be practically raw or at least not have reached the temperature necessary to destroy parasites.

Meat that is well cooked throughout always reaches from 60°-70° C. on the inside. It should be remembered that heat penetrates a large piece of meat slowly. For example, it requires 1½ hours in boiling water for the temperature to reach 62° C. in the interior of a piece of meat weighing 3½ pounds. Meat placed in a quick oven or broiled soon forms a hard coagulated and insulated coating that retains the juices, but retards the penetration of the heat.

Cooking softens the connective tissue and renders meat more tender. The bundles of fibrillæ are loosened from each other, the albumin is coagulated, the flavors are improved, and new flavors are developed, all of which enhances its digestibility.

Metchnikoff in his "new" hygiene dwells upon the great sanitary value of cooking. Perhaps no other single factor in preventive medicine protects us to an equal degree against infection. Metchnikoff believes that we should eat nothing in its raw state. This seems almost as extreme as the cult which proclaims the contrary.

One of the important functions in the preparation of food is to render it savory, tender, and appetizing. Foods that appear inviting

aid digestion by stimulating the secretion and flow of the digestive juices. Foods that are rendered soft and tender are more readily digested, but it should not be forgotten that the teeth need exercise to keep them in good condition. Tough meats may be pounded to separate the connective tissue bundles, or may be chopped or minced as an artificial aid to mastication, or may be steeped for several hours in fresh milk or sour milk, in which case the fibers are softened through the action of the bacteria and their enzymes. In the case of vegetables, cooking breaks open and softens the cellulose envelopes and fibers; the starch grains swell and burst, and the insoluble starch is converted into soluble starch or dextrine.

Fermentation is of great use in the preparation of foods. The best example is the leavening of bread. The yeast ferments the carbohydrates in the flour with the production of carbon dioxid and alcohol. The carbon dioxid renders the bread porous; the gas is held within the loaf on account of the glutenous property of the protein (gluten) in the flour. Fermentation is an adjunct in the preparation of many other foods and beverages, such as cheese, sauerkraut, vinegar, beer, wine, cider, etc.

The observations of Becker, Grove, and others concerning the heat of cooking are practical and important in the preparation of food. Exposure to steam at 60° to 70° C. for a long time has the advantage of cooking foods thoroughly throughout. This treatment prevents burning or the results of overheating; the juices are retained. The process requires little or no attention. Meat is thereby rendered tender and juicy, vegetables thoroughly soft, and the starch grains are all opened. A modification of this method is found in the fireless cookers now offered for sale in various forms. These devices consist simply of a well-insulated box. The food is first heated, then placed in suitable compartments, and a temperature above 70° C. maintained for many hours.

Certain precautions are advisable in the choice of pots and pans used in cooking. Brass and copper are not advisable, and if used must be kept scrupulously clean. Acid foods should not be cooked in copper vessels, and milk and saccharin substances should not be kept in copper containers on account of the possibility of the organic acids dissolving the copper. Tin, nickel, and aluminiumware are least objectionable. Enameled ware is entirely satisfactory, provided it does not contain lead.

Methods of Cooking.—Much depends upon the method of cooking. The principal methods in ordinary use are: roasting, broiling, boiling, frying, and stewing.

Roasting or broiling causes considerable shrinking, due mainly to loss of water. The heat coagulates the exterior of the meat and thus

prevents the further loss of juices and drying up. In order to obtain adequate heating of the meat throughout a large joint without burning and drying the exterior, it is necessary to baste it from time to time with hot melted fat. This also helps to form a protective coating.

In BOILING the meat is placed either in cold or hot water, depending upon the object desired. If it is desired to maintain the flavors within the mass, the meat should be plunged into boiling water. This quickly coagulates the albumins at the surface. If a rich broth is desired the meat should be placed in cold water and gradually heated. In this way the soluble albumins and extractives pass out into the surrounding water. The albumin of meat begins to coagulate at 134° F.; the connective tissue is changed to gelatin and dissolved above 160° F.

FRYING consists in placing meat or other substances into very hot fat, lard, or vegetable oil. This causes a speedy coagulation of the surface similar in all respects to that brought about in the process of boiling. The flavors and juices are thereby retained. If the fat is not very hot it will penetrate the tissues and cause the meat or other substance to become greasy and unpalatable. Fried substances are apt to be indigestible on account of the large amount of grease that adheres to and penetrates into them.

In STEWING the meat is cut into small pieces and placed in cold water, which then is heated slowly to about 180° F., at which the whole is kept for several hours. If heated above 180° F. the meat becomes tough, stringy, unpalatable, and of diminished digestibility.

CHAPTER II

ANIMAL FOODS: MILK

The animal foods used by man are not of great variety and source. They include the flesh and various organs of the herbivorous animals, swine, domestic and wild fowl, eggs, fish, shellfish, insects and their products (honey), milk, and milk products. The flesh of carnivorous animals, except that of fish, is unpalatable and, therefore, undesirable as a food.

The most important animal foods from the standpoint of the sanitarian are milk and meat.

MILK

Milk is responsible for more sickness and deaths than perhaps all other foods combined. There are several reasons for this: (1) bacteria grow well in milk; therefore, a very slight infection may produce widespread and serious results; (2) of all foodstuffs milk is the most difficult to obtain, handle, transport, and deliver in a clean, fresh, and satisfactory condition; (3) it is the most readily decomposable of all our foods; (4) finally, milk is the only standard article of diet obtained from animal sources consumed in its raw state.

The total milk production in the United States in 1911 was ten billion gallons. One-quarter of this is consumed as milk and the remaining three-quarters is used for butter and cheese. The average per capita consumption of milk in the United States is 0.6 of a pint daily. More milk is used in the North than in the South; very little in the tropics, and practically none at all in China, Japan, and some other countries. About 16 per cent. of the average dietary in the United States consists of milk and milk products.

Fresh milk products may be quite as dangerous as the milk from which they are made. Milk laws which ignore milk products are incomplete from the sanitary side, and will fail to accomplish their purpose from the economic side.

Milk is a perfect food for the suckling. It contains all the essential elements of a well-balanced diet for the adult, and at prevailing

prices it is one of the cheapest of the standard articles of diet. Furthermore, it is readily digestible and is capable of a great variety of modifications. The sanitarian, therefore, has every reason to encourage the use of pure milk as well as to discourage the use of impure milk.

Composition.—Milk is the secretion of the mammary gland. In composition it is exceedingly complex, consisting chiefly of water; several proteins in colloidal suspension; fats in emulsion; sugar, and a number of inorganic salts in solution; also enzymes, as well as antibodies, cells, gases, and other substances. Milk from all animals shows a general agreement in physical properties and composition, containing essentially the same ingredients, but exhibiting differences in the amounts of the several constituents.

In the fresh state milk is a yellowish white, opaque fluid. Cow's milk has a specific gravity of 1.027 to 1.035; it freezes at a temperature somewhat lower than the freezing point of water (—0.554° C.); the electrical conductivity is 43.8×10^{-4} for cow's milk, and 22.6×10^{-4} for human milk. In other words, 58 per cent. of the molecules in cow's milk and 26 per cent. in human milk are dissociated. The specific heat of milk containing 3.17 per cent. of fat is 0.9457. The coefficient of expansion is greater than that of water. Milk shows no maximum of density above 1° C.

Freshly drawn milk of carnivorous animals is, as a rule, acid in reaction. This is probably due to CO_2 and acid phosphates. Human milk and that of most of the herbivora are slightly alkaline; cow's milk has been described as amphoteric.

Under the microscope milk is found to contain fat globules and cells, as well as bacteria, débris, and other objects.

The gases dissolved in milk[1] are oxygen, nitrogen, and carbon dioxid (3 to 4 per cent. by volume). Oxygen and nitrogen are carried into milk mechanically from the air in the process of milking. Other substances found in milk, but in small quantities, are lecithin, cholesterin, citric acid, lactosin, orotic acid, and ammonia.

The composition of cow's milk may be understood from the scheme prepared by Lucius L. Van Slyke, given on page 496.

Proteins.—The three proteins constantly found in milk are casein, lactalbumin, and lactoglobulin. A trace of fibrin, mucin, and other proteins sometimes occurs.

The proteins in milk of a given species are quite constant both in composition and amount; it is, therefore, not necessary, as a rule, to make a special analysis for them. They may be estimated by subtracting the fat, sugar, and ash from the total solids.

Casein is a highly specialized protein found in the secretion of the milk glands of all mammals, but nowhere else in nature; it is a nucleo-

[1] When not otherwise specified in this section milk refers to cow's milk.

[Van Slyke]

Milk = 100.0
- Water = 87.1
- Solids = 12.9 (100.0)
  - Fat = 3.9
  - Solids not fat = 9.0 (12.9)
    - Nitrogen compounds = 3.2
      - Casein = 2.5
      - Albumin, etc. = 0.7 (3.2)
    - Milk sugar = 5.1
    - Ash (salts) = 0.7 (9.0)
- Gases
  - Carbon dioxid
  - Nitrogen
  - Oxygen

[Babcock]

Milk = 100.0
- Butter fat = 3.6
  - Olein
  - Palmitin
  - Stearin
  - Myristin
  - Butin (trace)
  - Butyrin
  - Caproin
  - Caprylin (trace)
  - Caprinin (trace)

  Glycerids of insoluble and nonvolatile acids 3.3

  Glycerids of soluble and volatile acids 0.3 (3.6)

  Fat 3.6
- Milk serum = 96.4 (100.0)
  - Casein 3.00
  - Albumin 0.60
  - Lactoglobulin, Galactin, Fibrin (trace) 0.20 (3.80)

  Containing nitrogen .. 3.8
  - Milk sugar 4.5
  - Citric acid 0.1
  - Potassium oxid 0.175
  - Sodium oxid 0.070
  - Calcium oxid 0.140
  - Magnesium oxid 0.017
  - Iron oxid 0.001
  - Sulphur trioxid 0.027
  - Phosphoric pentoxid ... 0.170
  - Chlorin 0.100 (0.7)

  Ash 0.7 (9.1)

  Solids not fat .. 9.1 (12.7)

Total solids .. 12.7

Water 87.3

100.0

albumin, and as such contains phosphorus. It is soluble in water, and by virtue of its property as an acid it forms soluble salts with alkalies. There are two series of casein salts, basic and neutral; solutions of the latter have a milky appearance. In milk, casein is found dissolved in the form of a neutral calcium salt, which accounts in part for the white opalescent appearance of milk. Casein exists in milk in the form of caseinogen, that is, casein in combination with calcium phosphate. The caseinogen is held in solution by the calcium phosphate. It is not coagulated by heat, but is precipitated by acids, for the reason that acids take the calcium from the calcium phosphate, and thus throw the casein out of solution as a curd. This flaky or lumpy precipitate is again soluble in lime water and dilute alkalies. Casein is also thrown out of solution by rennin.

Lactalbumin is very similar to the serum albumin of the blood, but it appears to differ from this in some particulars. It coagulates by heating to 70° C., but not with dilute acids, and is precipitated by a saturated solution of ammonium sulphate, but, like all other albumins, is not precipitated in a neutral solution of sodium chlorid and magnesium sulphate. Lactalbumin contains sulphur but no phosphorus. It is present in amounts varying from 0.2 to 0.8 per cent., but is much more abundant in colostrum.

Lactoglobulin occurs in milk in very small quantities, merely in traces, while colostrum is comparatively rich in this protein. It coagulates at 75° C., it is precipitated in the same way as serum globulin, and, like serum globulin, is insoluble in water, but is soluble to some extent in weak salt solution.

Fat.—The fat is suspended in the milk serum in the form of an emulsion. The droplets or globules vary in size. On the average they are smaller in milk from Holstein than from Jersey, Guernsey, or shorthorned breeds. Under the microscope some of the fat globules seem to have an albuminous membrane, but this interpretation is now questioned. The fat droplets are lighter than the milk serum, hence they rise on standing (gravity cream), or they may readily be separated by centrifugal force (centrifugal cream). Cream, or top milk, does not consist of fat alone, but contains all the constituents of the milk; it is simply milk rich in fat. Upon shaking the fat globules gradually coalesce into larger drops and lumps to form butter.

The first milk drawn from the udder is commonly poor in fat. This is known as "fore" milk. The middle portion contains about the average percentage of fat, and the last, known as "strippings," is always the richest in fat. The strippings may contain as much as 9 or 10 per cent. butter fat.

Heat increases the viscosity of milk, and hence hinders the rising of the fat drops; 68° C. is the critical temperature; if heated above this

point for any length of time the formation of the cream line is retarded or prevented. For this and other reasons the richness of milk, therefore, cannot always be judged by the depth of the cream layer.

Milk fat consists of a mixture of different neutral fats, the principal of which are olein, palmitin, and stearin. These are neutral triglycerids of the corresponding fatty acids. Besides these are found the triglycerids of miristic, butyric, and caprylic acids. The last two are volatile and give to butter its characteristic odor and flavor. The composition of the fat is subject to variation, depending upon racial or individual peculiarities, also upon the character of the food and other conditions.

The percentage of butter fat in milk has long been one of the standards by which milk is tested. The richness of milk gaged by the amount of fat it contains is more of an economic than a sanitary question. Milk with a low percentage of fat from Holstein cows is relatively just as nutritious a food as richer milk from Jersey and Guernsey cows; even skimmed milk containing little or no fat is a valuable food. The problem is one of honest labeling and the marketing of various grades at prices corresponding to their nutritive contents. When the standard for butter fat in milk is relatively low, say 3.25 per cent., it is a temptation for dairy men to remove the excess. This is a fraudulent practice which should not be countenanced. A high fat standard encourages the breeding of better cows; requires caution in their feeding and care, and puts a premium upon good dairy methods.

In normal milk the larger proportion of the fat droplets agglutinate into tiny clusters or masses. At a temperature of 65° C. or above these clusters are broken up and the globules are more homogeneously distributed throughout the liquid. When milk is subjected to a pressure of about 3,000 pounds at a temperature of about 75° C. the individual fat globules are broken up into fine particles, which remain as a uniform and permanent emulsion known as "homogenized milk." This process applied to cream increases its viscosity, so that cream containing 20 per cent. butter fat appears to have the body and richness of a 30 per cent. cream.

Researches of Heubner, Keller, and Czerny show that the fats and not the proteins are the cause of much of the digestive disturbances in infants. When the fat is excessive in amount the infant at first seems to thrive, but sooner or later loses weight and appetite, and shows other symptoms, associated with stools composed largely of fat soaps and of a pale gray, hard, and dry constituency. The alkaline bases are so largely drawn upon from the body to saponify the excessive amount of fat in the intestines, that a condition resembling acidosis may appear; furthermore, fermentative changes take place in the intestines and the "catastrophe" ensues.

Fat is the most variable constituent in milk. The amount varies with different animals, and even in the same animal from time to time.

Milk Sugar, or Lactose.—Milk sugar, or lactose ($C_{12}H_{22}O_{11}$), is peculiar to milk; it is found nowhere else in nature. Its formation is not understood. Commercially, milk sugar is obtained from whey as hard rhombic crystals, which have a slightly sweet taste and are soluble in six parts of cold water. Lactose is readily acted upon by microorganisms and reduced to glucose and galactose; the glucose is further changed to lactic acid. This is the common cause of sour milk (see The Fermentation of Milk, page 507).

Lactose, like glucose, reduces Fehling's solution when heated; it is dextrorotary. When heated above the boiling point of water it changes to a brownish color as a result of the formation of lactocaramel.

The amount of lactose in milk of any given species is remarkably constant.

Milk Standards.—Milk that meets standard requirements is not necessarily standard milk. The legal standards are minimum requirements and express inferiority, if anything. The standards are the lowest grades that the law will permit. There are, in fact, three standards by which milk should be judged: (1) the chemical standards; (2) bacteriological standards; (3) standards determined by inspection. All three are necessary for the satisfactory control of the milk supply.

The principal chemical standards are those for butter-fat and total solids. The legal requirements for the butter-fat and total solids in milk vary somewhat in different states, as shown by the following table:

LEGAL REQUIREMENTS

Fat per cent.		Solids not fat [1] per cent.	Total solids per cent.
3.0	Idaho	8.0	11.0
	California, Illinois, New Jersey,[1] New York,[1] Wisconsin	8.5	11.5
	Montana, North Dakota, Ohio, Porto Rico	9.0	12.0
	Iowa,[1] Michigan,[1] Oklahoma[1]	9.5	12.5
3.2	Oregon, Utah	9.0	12.2
3.25	Association of Official Agricultural Chemists, Connecticut, Georgia, Indiana, Kentucky, Maine, Missouri, North Carolina, South Dakota, Tennessee, Texas, Virginia	8.5	11.75
	Washington	8.75	12.00
3.35	Massachusetts[1]	8.8	12.15
3.5	Hawaii[1]	8.0	11.5
	District of Columbia, Maryland[1]	9.0	12.5
	Louisiana, New Hampshire	9.5	13.0

[1] These states marked do not directly specify the solids not fat. The figure given in such cases is the difference between the required total solids and the required fat.

It has been found an advantage to keep the butter-fat standard relatively high and the total solids at a minimum of 12 per cent. This allows 8.5 per cent. for solids not fat, such as the proteins, milk sugar, and inorganic salts. A 3.25 per cent. butter-fat and a 12 per cent. total solids is the minimum that should be allowed.

If the law recognizes a low standard for total solids, it permits manipulation of the milk, such, for example, as adding water. It also encourages the production of milk from inferior cows. High standards encourage good dairy methods, require good feed, and place a premium upon the better breeding of milch cows.

The determination of fats and total solids is used to detect skimming or watering; however, it is possible to skim milk or water it, within limits, without the possibility of detecting it through the fats and total solids.

If dependence is placed upon the total solids, mistakes may also occur. The total solids represent the proteins, fats, sugar, and inorganic salts. They may readily be tampered with. Thus sugar may be added to replace the cream that is taken off.

Ferments or "Life" in Milk.—Milk contains a large number of very active ferments or enzymes. These substances are the nearest approach to "life" that we know of in milk. Milk also possesses certain other properties common to blood and living tissues, but, while milk may properly be regarded as a vital fluid, it possesses none of the fundamental properties of life. In fact, milk begins to decay the moment it is drawn; ofttimes decomposition begins while the milk is still within the udder. It would, therefore, be more proper to regard milk as a dead fluid, in the same sense that shed blood is dead.

The ferments are believed to be important to the infant, and this importance has been emphasized especially since the introduction of pasteurization, for the reason that a high degree of heat destroys them. Some of the ferments in milk are normal constituents of that secretion, while others are produced by bacteria. Many tests have been devised to determine the kinds and activity of the ferments in milk. The tests most frequently and successfully used are those for catalases and reductases. The absence of ferments in milk indicates that it has been heated. The presence of certain ferments gives an indication of the age of the milk, the number of bacteria it contains, and also helps to distinguish between fresh normal milk and pathologically changed milk

The enzymes in milk are the following:

Galactase.—Galactase is a proteolytic ferment, similar to trypsin. It was found by Babcock and Russel to be abundant in separator slime. Ordinarily galactase by itself acts too slowly to cause any material change in the proteins in the short intervals which elapse between the withdrawal of the milk from the animal and its consumption as food.

Snyder claims that this enzyme probably assists digestion, in that when milk is used in a mixed diet the proteins have been found to be from 4 to 5 per cent. more digestible than when milk is omitted from the diet.

Lactokinase.—Hougardy has recently shown that milk contains a ferment or a kinase similar to enterokinase. Lactokinase has been found to accelerate the digestion of proteins by pancreatic juice. This property is destroyed by heating the milk at 73° to 75° C.

Lipase.—This fat-splitting ferment was found in milk by Marfan and Gillet. Human milk exhibits this property to a higher degree than cow's milk. The former has a lipolytic activity of 20 to 30 on Harriot's scale, while cow's milk shows an activity of only 6 to 8. Lipase withstands cold, but is destroyed by heating to 65° C.; it is monodialyzable and is held back by a porcelain filter. It probably hydrolyzes the higher fats of milk, at least to some extent, and may possibly account for a small part of the acidity of some milk.

Catalase.—Milk contains no true oxidases or oxidizing ferments proper (Kastle). It decomposes hydrogen peroxid and has the power of effecting the oxidation of a considerable number of easily oxidizable substances in the presence of hydrogen peroxid or ozonized oil of turpentine. In other words, milk contains catalase and peroxidase. Catalase is widely distributed among animals and plants. Jolles has pointed out that human milk decomposes five or six times as much hydrogen peroxid as cow's milk. Considerable importance has been attached to this difference, which has also been used to distinguish human milk from cow's milk. Little is known of the function of catalase. Hydrogen peroxid is probably formed in both animal and vegetable tissues during vital activities. The catalase would destroy it and thus prevent its accumulation in the cell, which otherwise would destroy its life.

Peroxidases.—Milk contains substances capable of inducing the oxidation of guaiacum and other readily oxidizable substances by means of hydrogen peroxid or ozonized oil of turpentine. These substances are known as peroxidases. The peroxidases are destroyed when milk is heated to 80° C. The color reactions for these ferments are a convenient test to determine whether milk has been heated beyond a certain temperature or not. The interpretation of this reaction must, however, be guarded, as Gillet and Kastle found that even normal fresh milks vary in the amount of peroxidases which they contain.

Reductases.—Raw milk possesses reducing properties; for example, it reduces Schardinger's reagent, which consists of a solution of methylene blue containing small amounts of formaldehyde.

Diastase (*Amylase*).—Bechamp in 1882 isolated from milk a ferment which liquefies starch and converts it into sugar as readily as

diastase. These observations have not been confirmed by recent investigation (Mora, Van De Velde, and Landtsheer, or Kastle).

Thermal Death Point of Milk Enzymes.—The influence of temperature on the activity of milk enzymes is very much like enzymes from other sources. All of this great group of substances stand in such intimate and close relation to the vital activities of the cell that all those conditions and influences which tend to destroy the one tend also to destroy the other. All of the bacteria in milk cannot be destroyed without rendering the ferments in milk inactive; but the non-spore-bearing bacteria can be killed without appreciable harm to the ferments, for in general the ferments have a higher thermal death point than such bacteria. The activity of ferments begins to be influenced at 60° C., and is seriously affected at 70° C.; at 80° C. they are destroyed. The non-spore-bearing bacteria are destroyed at 60° C. It is, therefore, possible to destroy all the serious infections in milk, so far as man is concerned, without influencing its "life," so far as the ferments are concerned. In fact, it has been shown that milk heated to 60° C. increases the activity of some of the ferments, notably the peroxidases.

ENZYMES IN MILK AND THEIR THERMAL DEATH POINTS[1]

Galactase—Proteolytic ferment	70° for 10 minutes retards its action. 76° for 10 minutes destroys its digestive power. (Babcock and Russell.) Not weakened at 60° for one hour. (von Freudenreich.) Withstands 65° for half an hour. (Hippius.)
Lactokinase—Accelerates pancreatic digestion	Destroys at 73° to 75° C. for half an hour. Enfeebled at 75° for 20 minutes. (Hongardy.)
Lipase—Fat-splitting ferment	Destroys at 70° C. (Harriot.) Destroys at 65° to 70° C. (Kastle and Loewenhart.) Withstands 60° for one hour. (Hippius.)
Catalase—Decomposes H_2O_2, etc	?
Peroxidase—Oxidizes guaiacum, etc.	Destroyed at 79° C. (Marfan.) Destroys at 76° C. (Hippius.)
Reductase—A reducing ferment	Existence is doubtful in Milk.
Diastase—Converts starch into sugar.	Probably does not exist in Milk. Diastasein saliva destroyed at 65° to 70° C.

[1] Compiled from Kastle.

"Leukocytes" in Milk.—A large number of cells are normally present in milk. These are not to be regarded as the result of inflammation, unless they have the characteristics of pus "cells." Those found in normal milk are, for the most part, degenerated epithelial cells. The number of cells in milk is greatly increased in the presence of garget; toward the end of lactation; on approaching calving time; during

periods of excitement, and various other factors. A leukocytic content of 500,000 or over to the cubic centimeter, especially in a mixed milk, is regarded by the Boston Board of Health as suggestive of some inflammatory condition of the udder, more particularly if associated with streptococci. Such milk is excluded until after satisfactory veterinary inspection of the herd.

Various methods have been proposed to count the number of cells in milk (see Microscopic Examination, page 525).

The Excretion of Drugs in Milk.—The following drugs taken by the mouth have been found in the milk of nursing women: aspirin, iodin, mercury (calomel), arsenious acid, potassium bromid, and probably also urotropin (hexamethylamin), salicylic acid, and salicylates, ether, antipyrin, bromids, and many others; the list is very long. It is probable that opium, all volatile oils, purgative salts, and rhubarb are excreted to a certain extent in the milk. It is well known how readily the flavor of cow's milk is affected by turnips, garlic, wild onions, moldy hay and grain, or damaged ensilage. Fermented distillery waste gives a bad flavor and may also cause the secretion of small quantities of alcohol in the milk. The importance of these facts is self-evident. Cows in pastures sometimes feed on poisonous weeds, and these poisons may pass into the milk. In the production of certified milk, cows are never allowed to graze, but are given carefully selected feed. Certain substances, as ensilage, when fed to cows, cause a laxative property to appear in the milk, and thus it is possible to affect the baby through the feed of the cow.

The Differences Between Cow's Milk and Woman's Milk.—The following table from Rotch summarizes the principal points of differences between cow's milk and human milk:

Woman's Milk Directly from the Breast	Cow's Milk, Freshly Milked
Reaction, amphoteric (more alkaline than acid)	Amphoteric (more acid than alkaline)
Water, 87 to 88 per cent	86 to 87 per cent.
Mineral matter, 0.20 per cent	0.70 per cent.
Total solids, 13 to 12 per cent	14 to 13 per cent.
Fats, 4.00 per cent. (relatively poor in volatile glycerids)	4.00 per cent. (relatively rich in volatile glycerids)
Milk sugar, 7.00 per cent	4.75 per cent.
Proteids, 1.50 per cent	3.50 per cent.
Caseinogen, ⅓ to ½ of the total proteids	2.66 per cent.
Whey-products, ⅔ to ½ of the total proteids	0.84 per cent.
Coagulable proteids, small proportionately	Large proportionately
Coagulation of proteids by acids and salts, with greater difficulty. Curds small and flocculent	With less difficulty. Curds large and tenacious
Coagulation of proteids by rennet, does not coagulate readily	Coagulates readily
Action of gastric juice, proteids precipitated but easily dissolved in excess of the gastric juice	Proteids precipitated but dissolved less readily

The differences between these two milks are greater than the table indicates. While cow's milk may be modified to approximate woman's milk in composition, it can never be just the same or just as good for infants.

Cow's milk is more opaque than woman's milk, although the latter may contain a greater percentage of fat. This is due to the opacity of the calcium-casein, which is present in greater proportion in cow's milk. Cow's milk is faintly acid or amphoteric when freshly drawn, but ordinarily is distinctly acid in reaction when consumed. Woman's milk is amphoteric or alkaline.

There is three times as much protein in cow's milk as in woman's milk. The reason for this is obvious, when we recall that the ratio of the growth of the calf to that of the infant is about as two to one. Furthermore, the protein in cow's milk consists chiefly of casein (3.02 per cent.) and little lactalbumin (0.53 per cent.), while woman's milk contains 0.59 per cent. of casein and 1.23 per cent. lactalbumin. The sugar in the two milks varies greatly in amount, but not in kind. Cow's milk contains almost four times the amount of inorganic salts compared to woman's milk. Of more importance, the salts in cow's milk consist mainly of the calcium and magnesium, while those in woman's milk consist mainly of potassium and sodium bases. These differences have an important bearing upon infant metabolism. There is no great difference in the average amount of fat in the two milks; however, both in woman's milk and in cow's milk the fat is the most variable constituent.

The curd from cow's milk is usually tougher and in larger masses than that in woman's milk. There are also differences in the antibodies, ferments, etc.

Classification of Milk.—From a public health standpoint there are only two kinds of milk—good and bad. We are, however, confronted with a complex situation which has resulted in various schemes for grading milk according to its sanitary quality and its nutritive value. Perhaps the most practical classification has been advanced by the government, viz.: (1) certified milk; (2) inspected milk; (3) market milk. A fourth grade known as "cooking milk" or "milk not suitable for drinking purposes" has been proposed, but has not met with favor except from the economic standpoint in large cities.

There is a growing tendency to classify all milk into raw and pasteurized. This is the most satisfactory classification from a sanitary standpoint.

Certified Milk.—The term "certified milk" was coined by Dr. Henry L. Coit of Newark, N. J., who in 1892 formulated a plan for the production of clean, fresh, pure milk under the auspices of a medical milk commission. The term "certified milk," then, is milk of the highest

quality, of uniform composition, obtained by cleanly methods from healthy cows under the special supervision of a medical milk commission.

The use of the term "certified milk" should be limited to milk produced in accordance with the requirements of the American Association of Medical Milk Commissions.[1] The first requisite in the production of certified milk is to enlist the coöperation of a trustworthy dairyman who is willing to enter into a contract with the medical milk commission. In accordance with the terms of this contract, the dairyman binds himself to comply with the specifications set forth and in return his milk is certified.

The dairies are subjected to periodic inspections, and the milk to frequent analyses. The cows producing certified milk must be free from tuberculosis, as shown by the tuberculin test and physical examination by a qualified veterinarian, and from all other communicable disease, and from all diseases and conditions whatsoever likely to deteriorate the milk. They must be housed in clean, properly ventilated stables of sanitary construction, and must be kept clean and properly fed and cared for. All persons who come in contact with the milk must exercise scrupulous cleanliness, and must not harbor the germs of typhoid fever, tuberculosis, diphtheria, or other infections liable to be conveyed to the milk. Milk must be drawn under all precautions necessary to avoid contamination, and must be immediately cooled, placed in sterilized bottles, and kept at a temperature not exceeding 50° F. until delivered to the consumer. Pure water, as determined by chemical and bacteriological examination, is to be provided for use throughout the dairy farm and dairy. Certified milk should not contain more than 10,000 bacteria per cubic centimeter, and should not be more than twenty-four hours old when delivered.

Inspected Milk.—This term should be limited to clean, fresh milk from healthy cows, as determined by the tuberculin test and physical examination by a qualified veterinarian. The cows are to be fed, watered, housed, and milked under good conditions, but not necessarily equal to those prescribed in the production of certified milk. Scrupulous cleanliness must be exercised and particular care be taken that persons having communicable infections do not come into contact with the milk. This milk must be delivered in sterilized containers, and kept at a temperature not exceeding 50° F. until it reaches the consumer. There should not be more than 100,000 bacteria per cubic centimeter of inspected milk.

Market Milk.—All milk that is not certified or inspected in accordance with the above definitions, and all milk that is of unknown origin, is classed as "market milk," and should be pasteurized.

[1] See annual reports of this Association.

The Decomposition of Milk.—Milk spoils in various ways as the result of bacterial growth; the kind of decomposition depending upon the kind of bacteria which predominate. Milk, as a rule, ferments, but sometimes it putrefies. In the former case the main change takes place in the carbohydrates; in the latter the proteins are broken down. The fermentation, known as the souring of milk, is accompanied by an acid reaction and a precipitation of the casein. Putrid milk turns alkaline and bitter, owing to the formation of peptones. Sour milk is regarded as the normal form of decomposition, because it is the usual change and is not harmful. Putrid milk is believed at times to contain toxic substances; it is at least suspicious.

Fig. 68.—Unsanitary Surroundings of a Cow Barn.

Sour Milk—Lactic Acid Fermentation.—Milk curdles or sours when the soluble caseinogen is thrown out of solution and precipitated as casein. The caseinogen exists in milk as a complex molecule containing calcium phosphate loosely bound to it; it also contains calcium as part of the molecular complex. The formula may be expressed thus:

$$Ca_3(PO_4)_2 \text{ (Ca. Caseinogen)}$$

The casein is held in solution (colloidal suspension) by the calcium phosphate and other soluble salts of calcium. Any chemical reaction that removes the calcium phosphate from this combination causes a precipitation of the caseinogen as casein. The casein may be precipitated by various substances, such as rennin or acids. In the normal curdling or souring of milk the casein is precipitated by lactic acid

produced through the action of bacteria upon lactose. The lactic acid results from hydrolysis of the lactose as follows:

$$C_{12}H_{22}O_{11}+H_2O=C_6H_{12}O_6+C_6H_{12}O_6$$
$$(\text{lactose})=(\text{galactose})+(\text{glucose})$$
$$C_6H_{12}O_6=2C_3H_6O_3$$
$$(\text{glucose})=(\text{lactic acid})$$

The bacteria usually concerned in the souring of milk are: *B. acidi lactici* of Hueppe, *B. lactis acidi* of Leichmann, *Streptococcus lactis* of Kruse, *B. bulgaricus* of Metchnikoff, *B. aerogenes capsulatus* of Welch, *B. coli,* and a great number of other microorganisms capable of fermenting sugar with the production of acid.

Sour milk, obtained from clean milk, is a beneficial food. It contains myriads of lactic acid bacteria. Metchnikoff has recently called attention to the importance of a normal lactic acid flora in the large intestines, which inhibits putrefactive processes and thereby stands guard against autointoxication. He recommends the use of certain bacteria in sour milk, especially *B. bulgaricus.* It is a fallacy, however, to suppose that the flora of the large intestines may be materially influenced through ingestion of these bacteria by the mouth, even when taken in enormous numbers, as in sour milk. Perhaps the best way to influence the bacteria of the large intestines is through diet. A protein diet favors a putrefaction flora; a carbohydrate diet a normal flora. Kendall, in his work on intestinal bacteriology, has shown that carbohydrates spare proteins; that is, bacteria do not ordinarily break down protein in the presence of carbohydrates.

Putrid Milk—Alkaline Putrefaction.—When boiled milk is allowed to stand at room temperature, it gradually acquires an alkaline reaction,[1] a bitter taste, and finally curdles, yielding a soft, slimy curd. On further standing this curd is peptonized to form a somewhat clear fluid, and if these putrefactive changes are allowed to proceed for a sufficient length of time a semi-transparent liquid is obtained, having no resemblance to milk. In this form of decomposition the main change occurs in the protein constituent of the milk. The putrefactive changes of milk are undesirable and are believed sometimes to be dangerous, in that toxic substances resembling "ptomains" may be produced. The principal cause of putrefaction in milk is the spore-bearing group of bacilli, belonging to and resembling the hay bacillus and also the anaerobes.

Slimy or Ropey Milk.—Under some circumstances certain mucilaginous substances develop in milk through abnormal fermentation. Slimy milk has been obtained of such viscosity that it could be drawn

[1] Schorer found that such milk becomes less acid but seldom becomes actually alkaline in reaction.

out into threads ten feet in length, and of such thinness as to be scarcely visible. In Norway such milk is esteemed a delicacy; in this country, however, it is objectionable. From a health standpoint ropey milk is not injurious unless it is slimy as a result of mucopurulent materials caused by diseased conditions in the mammary glands. The bacteria which produce ropey milk are widely distributed in nature. Of these *B. lactis viscosis* (Adametz) is the commonest organism found in Europe, and a similar organism occurs in this country. *B. lactis viscosis* is very hardy; it may find its way into the milk through the water supply of the dairy, and then become widely diffused and difficult to trace. It is sometimes very troublesome, but may be eradicated through cleanliness. Sometimes it is necessary to resort to disinfection. Other organisms producing sliminess in milk are the *Micrococcus freudenreichii,* two forms of streptococci, and certain of the lactic acid bacteria.

Alcoholic Fermentation of Milk.—This is an abnormal fermentation which sometimes occurs as a result of yeasts, aided in their action by certain species of bacteria. Alcoholic fermentation of milk seldom occurs spontaneously, but may be induced by direct inoculations with certain ferments, such as those employed in the production of kumyss and kefir.

Kumyss was originally made from mare's milk; is now made from cow's milk by the addition of cane sugar and yeast. *Kefir* is a similar beverage, originating in the Caucasus, where the fermentation is carried out in leather bottles and is started by means of "kefir grains" which contain yeast and various microorganisms.

Bitter Milk.—Freshly drawn milk sometimes has a bitter taste; in other instances milk acquires such a taste on standing a few hours. The former is due to improperly feeding the cow with such herbs as lupines, wormwood, raw Swedish turnips, cabbages, etc. The latter case is due to the growth of certain bacteria in the milk after it is drawn. The condition is undesirable, and sometimes causes much trouble for the dairyman, but it has no particular sanitary significance. According to Conn, it is a micrococcus, and according to Weigmann a bacillus, that has the power of ruining the taste of freshly drawn milk in a few hours. This condition should be distinguished from the bitter taste of putrid milk above noted.

Colored Milk.—Blue milk is usually due to the *Bacillus cyanogenes.* Such milk is apparently harmless. Red milk may be due to the presence of blood coming from an injury, or acute infection of the udder. Sometimes it results from the feeding of the cow on plants containing red pigment, such as the madder root. A red color may also be produced by the *Bacillus erythrogenes, B. prodigiosus,* and sarcinæ. Red milk caused through the agency of bacteria is without sanitary significance.

Adulterations of Milk.—*Skimming.*—The removal of part or all of the cream and selling the remaining fluid as whole milk is an economic fraud, and has no reference to health, except that the milk is correspondingly lowered in nutritive value.

Watering.—The practice of watering is not nearly so frequent as formerly. If the water be pure it must be regarded more as a fraud than a health problem. The addition of water to milk lowers its specific gravity, raises its freezing point, and lowers its index of refraction and also its viscosity.

Thickening agents, such as the use of chalk, calves' brain, and glycerin, have never been common practices. Gelatin or lime is sometimes used to thicken cream. Cream may also be thickened by homogenizing it. *Coloring matter* is sometimes added with the object of concealing skimming or watering or to make the milk look richer. Annato, a vegetable dye, is most commonly used; orange and yellow azo coal-tar are also used. *Alkalies,* such as sodium carbonate or bicarbonate, are occasionally added to milk to reduce its acidity or to improve its taste or to delay curdling. *Sweet substances,* such as saccharin or sugar, are occasionally added to milk, either to raise the specific gravity and thus disguise watering, or to disguise the sour taste of milk just on the turn.

Chemical Preservatives.—Chemical preservatives, such as borax and boracic acid, salicylic acid, benzoic acid and benzoates, potassium bichromate, peroxid of hydrogen, fluorids, formaldehyde, and others, have from time to time been used in milk. The practice of adding any chemical preservative to milk meets with the unqualified disapproval of the sanitarian. Almost all countries prohibit the use of such foreign substances. The only proper preservatives for milk are cleanliness and cold.

Dirty Milk—The Dirt Test.—Practically all milk contains more or less dirt. For the most part, this dirt consists of cow feces. The presence of dirt may best be determined by filtering a pint of milk through a little disk of absorbent cotton. This produces a stain varying in intensity from a yellowish to a brownish or black spot. A Gooch crucible, a Lorenz apparatus, or simply an ordinary funnel may be used to filter the milk. Warm milk filters much more readily than cold milk. This simple test is one of the most practical of the routine tests used for the public health control of milk supplies. The intensity of the stain and the amount of deposit upon the cotton is a tell-tale which appeals strongly to farmers and dairymen, as well as to consumers. It is a good practice to send these disks of cotton, with a letter, to the farmer, showing him the amount of dirt contained in his milk.

Bacteria in Milk.—As a rule, milk contains relatively and actually

more bacteria than any other article of diet. Milk may, in fact, contain more bacteria than any other known substance; it frequently contains many more bacteria than are found in sewage. Mere numbers, however, need not alarm us, for it is the kind that most concerns us. By universal consent, however, milk containing an excessive number of miscellaneous bacteria is not suitable for infant feeding. Were milk a transparent food the enormous growth of microorganisms present in average market milk would be plainly visible to the naked eye.

The bacteria get into the milk from a number of different sources. Some of them are in the milk before it leaves the udder. They grow up the milk ducts into the milk cistern; hence, the fore-milk contains more than the mid-milk or strippings. It is practically impossible to

FIG. 69.—CONDITIONS UNDER WHICH IT IS DIFFICULT TO CLEANSE AND DISINFECT MILK BOTTLES AND MILK PAILS.

obtain sterile milk directly from the teat in any large quantity. As soon as the milk leaves the teat it receives additional contamination from all objects with which it comes in contact, as the hands, the pail, the dust in the air, etc. Most bacteria get into milk with the dirt that falls from the belly and udder of the cow during milking.

The number of bacteria in milk increases every time it is handled or exposed in any way. Separator milk contains more bacteria than the original milk. The same is true of filtered milk. This is due to the fact that, while some of the visible dirt in the milk is taken out, the particles are broken up and the bacteria disperse throughout the fluid.

For the most part bacteria do not pass a healthy udder. However, we can place no trust in the filtering ability of the mammary

gland. It is known that the virus of foot-and-mouth disease, which is ultramicroscopic, and the virus of malta fever (*Micrococcus melitensis*), and also the virus of milk sickness are almost constantly found in the milk of affected animals. On the other hand, tubercle bacilli do not pass the mammary gland unless there is tuberculosis of the udder.

The bacteria in milk are not equally distributed throughout the fluid. There are more bacteria in cream than in the underlying skim milk—particularly in gravity cream. As the cream rises it mechanically carries the bacteria along with it, very much as a snowstorm sweeps the atmosphere. Milk formulæ for infant feeding are often made of top milk, which, however, may contain 5 to 100 times the number of bacteria per cubic centimeter found in the whole milk. In twenty-six samples of milk Anderson found the gravity cream contained about four times as many bacteria as the sediment layer, and about one-third as many as the whole milk. Schorer found that the cream from milk of high bacterial count contained several thousand times as many bacteria as the underlying skim milk.

Certified milk should not contain over 10,000 bacteria per c. c.; inspected milk not over 100,000, and market milk not over 500,000. New York has placed the limit at 1,000,000 per c. c. Even this standard, however, has not been rigidly enforced. Boston has a standard of 500,000; Rochester 100,000.

In Washington in 1908 the average bacterial count of the market milk was 22,000,000 per c. c., as found in many hundreds of samples of the city supply. In 1907 the average was reduced to 11,000,000.

Excessive numbers of bacteria in milk indicate that it is dirty, old, or warm. Any one or any combination of these factors favors a rapid growth and multiplication of the bacteria in milk.

Methods for determining the number and kind of bacteria in milk will be found on page 523.

The Germicidal Property of Milk.—The so-called germicidal property of milk has been much misunderstood. Judged by the number of colonies that develop upon agar plates, the bacteria in milk first diminish, then increase in number. This occurs only in raw milk during the first 8 or 12 hours after it is drawn. Although the bacteria seemingly decrease in numbers, they never entirely disappear. After this initial decrease there is a continuous and rapid increase, until the milk contains almost infinite numbers in each cubic centimeter. This power of milk to restrain the development of bacteria lasts from 6 to 24 hours, depending upon the temperature at which the milk is kept. When the milk is kept warm, 37° C., the decrease is pronounced within the first 8 or 10 hours; after this the milk has entirely lost its restraining action. When the milk is kept cool, 15° C., the decrease is less marked but more prolonged.

The decrease in the number of bacteria is largely apparent, being due, at least in part, to agglutination; that is, the bacteria are not killed, they are simply grouped in clusters; this is proven by the fact that these clusters may be shaken asunder. The germicidal action of milk is specific; at most, is feeble, and is destroyed if the milk is heated above 80° C. It varies in different animals, and in the milk from the same animal at different times. It cannot take the place of cleanliness and ice, but may be taken advantage of in good dairy methods. It is true that bacteria develop more quickly in heated milk than raw milk, provided the raw milk is fresh; it should be remembered, however, that milk that is a day old no longer possesses this restraining action. The germicidal property is, therefore, ordinarily absent in market milk.

Diseases Spread by Milk.—The diseases most commonly conveyed through milk are: tuberculosis, typhoid fever, diphtheria, scarlet fever, septic sore throat, malta fever, foot-and-mouth disease, and milk sickness, also some of the summer complaints of children, and the diarrheal and dysenteric diseases of adults, which are often referable to infected milk. When all the facts are brought together they make a strong indictment against milk. Thus, during the five years, 1907-11, there were five milk-borne outbreaks in Boston, causing a total of over 4,000 cases of sickness.

Year	Milk-borne Epidemics in Greater Boston	Cases
1907	Diphtheria	72
1907	Scarlet fever	717
1908	Typhoid fever, about	400
1910	Scarlet fever, over	842
1911	"Septic sore throat," over	2,065
		4,096

As a rule, milk becomes infected from human sources, sometimes on the farm, sometimes at the dairy, sometimes in transportation, and occasionally in the household. Sometimes the milk becomes infected as a result of disease of the cow, as in the case of bovine tuberculosis, malta fever, foot-and-mouth disease, streptococci, etc.

In addition to the specific diseases, milk may be injurious as a result of other causes. Thus, Le Blanc has pointed out that the milk of cows in heat may cause gastrointestinal disturbances. The toxic effects of milk and milk products of nymphomanous cows are even more marked. Milk should not be used within fifteen days of parturition. The requirement for certified milk is placed at thirty days before and fifteen days after. Such milk is apt to produce diarrhea, colic, and

other digestive disturbances. Milk may further be harmful as a result of such diseases as mastitis or garget, gastroenteritis, septic and febrile conditions of the cow. Recently it has been shown that contagious abortion of cows is due to the *Bacillus abortus*, which may contaminate milk; it is pathogenic for many animals, probably including man. Schroeder and Cotton found this bacillus in 8 out of 27 samples of market milk tested. All such milk should be excluded or pasteurized.

Tuberculosis.—Bovine tubercle bacilli get into milk either directly as a result of tuberculosis of the udder, which occurs in from 1 to 2 per cent. of all tubercular cows, or indirectly through cow manure. In the latter case the tubercle bacilli are coughed up, swallowed, and passed in the feces. Practically all market milk contains cow feces. Occasionally milk contains tubercle bacilli of the human type from human sources. Tuberculosis in cattle is very prevalent. The "milk" from a tuberculous udder, when examined under the microscope, may contain as many tubercle bacilli as are ordinarily found in tuberculous sputum. The milk from a tuberculous udder of one cow may contain sufficient bacilli to infect the milk of 25 or 30 cows. In one case Ostertag found that 0.001 c. c. of the secretion from a tuberculous udder was sufficient to cause tuberculosis in a guinea pig. In such a case a child would receive an enormous dose in a gill.

Tonney examined the market milk of Chicago in 1910 for the presence of tubercle bacilli. In 10.5 per cent. of 144 samples of raw milk he found tubercle bacilli in sufficient numbers to infect guinea pigs. Of 19 samples of pasteurized milk examined none contained tubercle bacilli.

Hess in 1909 examined 107 samples of market milk in New York City, with the result that 17 of them, or 16 per cent., were found to contain tubercle bacilli.

Anderson examined 223 samples taken in the city of Washington, and reported 16, or 6.72 per cent., as positive. The tests made by the Bureau of Animal Industry of the milk in Washington disclosed 7.7 per cent. infected. Goler reports about 5 per cent. of the milk supply of Rochester, N. Y., infected.

To sum up, we have evidence from four typical American cities. A total of 551 samples of milk have been examined, in which tubercle bacilli were found in 46, making a percentage of 8.3. This may be taken as the average percentage for the entire country.

Professor Delepine found that the milk sent by rail to Manchester from 272 farms contained tubercle bacilli from 26, or 9.5 per cent. Wherever these investigations have been carried out similar and sometimes higher results have been obtained, both in Europe and in this country. It is believed that the figures are an underestimate, for the methods used in the laboratory are not sufficiently delicate to detect

a few tubercle bacilli in milk. Unless these microorganisms are present in considerable numbers, they are apt to escape detection. In any event, it is clear that the common market milk furnished all large cities and probably most small towns very often contains tubercle bacilli.

The frequency with which tubercle bacilli are found in butter is shown in a table collected by Swithinbank and Newman.[1] Of 498 samples tested from different sources, 76, or 15.2 per cent., contained tubercle bacilli.

Schroeder and Cotton[2] have found that living tubercle bacilli will retain their infective properties for at least 160 days in salted butter when kept without ice in a house cellar.

Mohler, Washburn, and Doane found tubercle bacilli to live a year and more in cheese 220 days old. In these experiments the cheese was purposely infected and fed or inoculated into guinea-pigs at various times.

The relation of bovine tuberculosis to man is considered on page 124.

Typhoid Fever.—Of milk-borne epidemics, typhoid fever takes the lead. Typhoid bacilli may swarm in milk without altering its taste, odor, or appearance. In Washington 10 per cent. of all the cases of typhoid fever during the four years 1907-10 were traced to milk. The milk may become infected by a convalescent, a carrier, or a missed case.

Bolduan estimates that from 300 to 400 cases of typhoid fever each year come in contact with the milk supplied New York City. He further states that "the startling total of 90 to 120 typhoid carriers now probably menace the milk supply of this city." This estimate is based upon the fact that about 200,000 persons come into more or less contact with the milk from over 40,000 dairy farms (see Typhoid Fever, page 89).

Scarlet Fever.—Milk-borne outbreaks of scarlet fever are sometimes extensive and serious. The milk is practically always infected from human sources. There is a suspicion, however, that sometimes streptococcal infections of the cow may reproduce a disease resembling scarlet fever in man (see Scarlet Fever, page 161).

Diphtheria.—Diphtheria bacilli in milk usually come from human sources, either cases or carriers. In a few instances ulcers upon the teat of the cow have become infected with diphtheria, and the bacilli are thus transferred to the milk. Such an occurrence, however, is unusual. As a rule, diphtheria outbreaks caused by infected milk are more limited both as to numbers and area than milk-borne outbreaks of typhoid or scarlet fever (see Diphtheria, page 146).

Septic Sore Throat.—The first milk-borne outbreak of "septic

[1] *Bacteriology of Milk*, p. 221.
[2] *Bureau of Animal Industry Cir. No. 153*, p. 38.

sore throat" recognized in this country occurred in and about Boston in May, 1911. Since then similar outbreaks have occurred in Baltimore, Concord, N. H., Chicago, and elsewhere. The infection is spreading. The Boston outbreak was carefully studied by Winslow and is so instructive that a brief account of it is given below.

Septic sore throat due to infected milk is well known in Great Britain. Swithinbank and Newman state that a year never goes by in which there are not outbreaks of sore throat or tonsillitis due to milk or cream. These infections appear to be due to a streptococcus, several varieties having been isolated both from the milk and the throats of the patients. It is assumed that the infection usually gets into the milk from human sources, although it is suspected that streptococci eliminated by diseased udders may be responsible for some outbreaks.

The disease often presents a severe clinical type and may result in death. Apparently it is not readily communicable from person to person. The inflammation and swelling of the lymphoid structures of the throat and of the mucous membranes are more severe than ordinarily; edema is a feature, and many cases present pseudomembranous formation and other indications of a virulent infection. There is a sharp febrile reaction, prostration, and sometimes delirium. The duration of the disease may be prolonged, and complications occur in about one-quarter of the cases. These consist mostly of enlarged regional lymph nodes, which may suppurate; abscesses, arthritis, endocarditis, peritonitis, erysipelas, pneumonia, pyemia, acute nephritis, otitis, and other sequelæ indicating the invasion of the blood with a virulent streptococcus.

The Boston outbreak in 1911 was characterized by its extraordinary virulence and comparative immunity of children, and high mortality among the aged and infirm. In this outbreak there were over 2,000 cases with about 48 deaths. One of the features of special interest was that the milk incriminated had always been a particularly clean, fresh, and satisfactory supply. It was obtained from tuberculin-tested cows under veterinary supervision, and the milk itself subjected to frequent chemical and bacteriological tests. The milk was bottled at the dairy, the bottles were sterilized, and many extra precautions were taken to ensure its cleanliness. For 28 years not a breath of suspicion was attached to this milk until this catastrophe occurred. It emphasizes the lesson that raw milk is apt to be dangerous milk, and our only protection against these particular dangers is through pasteurization.[1]

Milk Sickness.—Slows or trembles is a peculiar disease found in the central part of the United States. As forests are cleared and pastures fenced the disease becomes less frequent. It is still met with

[1] For a more detailed study of this and other milk-borne outbreaks see *The Milk Question,* by M. J. Rosenau.

in the valley of the Pecos River, New Mexico, in parts of Tennessee and North Carolina. The virus is communicated to man and is frequently fatal. Nancy Hanks, the mother of Lincoln, died from the disease in 1818 after an illness of a week. Little is known of the cause of milk sickness. Jordan and Harris have found a bacillus associated with the disease which they have called the *Bacillus lactis morbi.*

Milk sickness is an acute non-febrile disease due to the ingestion of milk, milk products, or the flesh of animals suffering from a disease known as trembles. The disease is characterized by great depression, persistent vomiting, obstinate constipation, and high mortality.

Malta Fever.—Malta fever is a disease primarily of goats; secondarily of man. The infection is transmitted from goats to man through milk containing the *Micrococcus melitensis* (see page 288).

Foot-and-mouth Disease.—Foot-and-mouth disease is an infection primarily of cattle and secondarily of man. It is caused by a filterable virus, and is noteworthy for being the first ultramicroscopic virus discovered by Loeffler and Froesch in 1898. The infection is transmitted to man through the ingestion of raw milk, buttermilk, cheese, or whey from diseased cows. Children are not infrequently infected by drinking unboiled milk when the disease is prevalent in the neighborhood. In man the disease is mild; the symptoms resemble those observed in animals; there is fever, sometimes vomiting, painful swallowing, heat and dryness of the mouth, followed by an eruption of vesicles in the buccal and mucous membranes, and very rarely by similar ones on the fingers. The vesicles are about the size of a pea; they soon break, leaving small erosions, which rapidly heal. The disease is seldom fatal except occasionally in very weak children.

The Character of Milk-borne Epidemics.—Milk-borne epidemics usually have an explosive onset, rise to a peak, and decline gradually. The character of the curve depends upon the amount of infection in the milk, and the manner of its distribution, the number of persons who drink it, and other factors. If the infection in the milk is dilute or attenuated, the disease crops out among a few susceptible persons who drink it. If the infection is concentrated and the milk is widely used, the curve of the outbreak will have the steeple-like character of a water-borne epidemic. The length of the epidemic varies with the period of incubation of the disease and with the length of time the milk is infected. The number of people involved may vary from a few to a hundred or several thousand. Only a single bottle of milk may be infected, and thus convey the disease to only one person; on the other hand, many gallons of mixed dairy milk may become infected and produce disease in many hundred persons. As a rule, milk outbreaks last a comparatively short time, and extend over a circumscribed area, as the disease follows the milk route. At first the disease occurs almost

exclusively among users of the infected milk. Afterward secondary cases may occur.

The disease shows a special incidence among milk drinkers. It is interesting to note that sometimes only one person of a number living in the same house is attacked, and such a one is a person who drinks the milk raw.

Milk-borne diseases attack those living under the best sanitary conditions. The reason for this is that such people drink milk more freely than the poor. Milk outbreaks among the well-to-do are unnecessary tragedies to the sanitarian.

Most milk outbreaks show a greater incidence of the disease among women and children, who are usually credited with drinking more milk

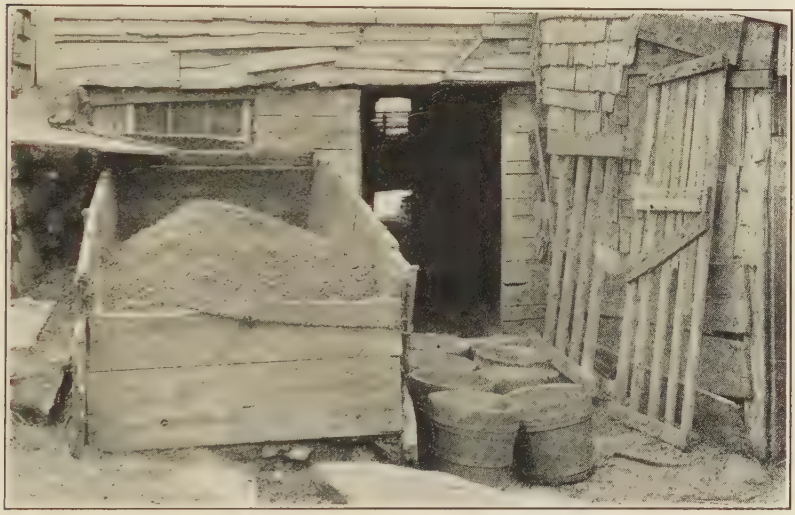


Fig. 70.—A Dark, Poorly Ventilated Cow Shed, Difficult to Keep Clean.

than men. There is apt to be a short period of incubation, probably on account of the concentration and large amount of the infection; however, the disease often runs a mild course. Multiple cases occur simultaneously in the same house. Such an occurrence is very suggestive to the epidemiologist, and frequently gives him the first hint of an impending milk epidemic.

Fresh Milk Products.—Cream, butter, buttermilk, ice-cream, sour milk, fresh cheese, and other milk products may convey all the infections contained in the original milk from which they are prepared. It is known that tubercle bacilli pass into butter and may live there for months. It has also been demonstrated that infected cream may be the cause of typhoid fever, septic sore throat, and without doubt diphtheria, scarlet fever, and other milk-borne diseases.

Milk products are frequently made from the left-over milk or milk otherwise unsalable. This may be controlled by an efficient system of inspection.

The infections in fresh milk products may be guarded against by pasteurization. It is comparatively easy to pasteurize cream, for the reason that it may be heated to a higher temperature than is the case with milk without materially altering its physical properties.

For so-called tyrotoxicon poisoning due to cheese see section on Ptomains.

Inspection.—An efficient inspection service is a preventive measure that strikes at the root of the milk problem. A good inspection service is expensive, but is worth its cost in providing cleaner and better milk. Inspection has its limitations, for it cannot see bacillus carriers, mild cases of disease, and cannot be on hand at all places at all times. No system of inspection can be so perfect as to insure milk free from infection at all times.

A competent system of inspection will help the farmer very much with his problems, and the educational value of such a system is one of its best features. The score-card system is an essential element in a successful inspection service.

Inspection is particularly helpful in tracing the source of infected milk and preventing recurrences. Another important element in any inspection system is the license or permit. All persons producing or handling milk should obtain a license, which should be issued only after the person has demonstrated his capacity to handle milk in a safe and cleanly manner.

Pasteurization.—Pasteurization as applied to milk consists in heating it for a short period of time at a temperature below the boiling point, followed by rapid chilling. In the language of the kitchen, pasteurization means parboiling. To the sanitarian pasteurization has but one object, viz., the destruction of bacteria.

Milk heated to 60° C. and held at that temperature for 20 minutes will kill the viruses of tuberculosis, typhoid fever, scarlet fever, diphtheria, malta fever, dysentery, foot-and-mouth disease; this time and temperature will also kill streptococci, staphylococci, and practically all non-spore-bearing microorganisms pathogenic for man. To provide a factor of safety it is advisable in commercial practice to heat milk to 65° C. for a period of 30 or 45 minutes. Heating milk to this temperature does not alter its taste, odor, or digestibility, does not interfere with its food value, and has the great advantage of preventing much sickness and saving many lives.

Pasteurization is not the ideal, but only a temporary, expedient. It is the simplest, cheapest, least objectionable, and most trustworthy method of rendering infected milk safe. Pasteurization, however, can-

not atone for filth and should not be used as a redemption process. A pure milk is better than a purified milk; however, no one should drink raw milk that cannot be guaranteed by the health officer as safe and free from danger. Only certified milk or milk of equally high character can be regarded as reasonably safe and satisfactory without pasteurization. Less than 1 per cent. of all the milk found upon the market comes within the honor class.

Pasteurized milk must be handled at least as carefully as raw milk. It should be bottled by machinery immediately following the process, kept cold, and delivered promptly. Pasteurized milk sours as a result of acid fermentation, just as raw milk does. In other words, the temperatures recommended do not destroy nature's danger signal—the lactic acid bacteria.

Pasteurization is not proposed as a substitute for inspection, but as an adjunct to inspection. Inspection gives us cleaner and better, but not necessarily safe, milk. Pasteurization eliminates the dangers inspection cannot see. The combination of inspection and pasteurization corresponds in all respects to modern methods of obtaining a safe water supply for a large city. The watershed, through inspection, is kept clean, but the water is filtered or purified before it is given to the consumer.

There can be no more objection to the heating of milk for the use of adults and children above the age of three years than there is to the cooking of meat. Infants should receive breast milk. When this is not possible they should have the best, freshest cow's milk that can be obtained. Whether such milk is to be pasteurized, modified, or otherwise treated will vary with circumstances.

Much has been said concerning the relation of scurvy and rickets to pasteurized milk. This is still a disputed point, but the evidence seems clear to me that these two diseases bear no relation whatever to the heating of the milk. Scurvy may readily be prevented by the use of a little orange juice, pineapple juice, or the juice of other fresh fruits. Rickets is a disease of defective alimentation, which cannot be laid to the door of pasteurization. Pediatricians now almost unanimously recommend pasteurization, particularly in the summer time, especially for those infants who must depend upon ordinary market milk or milk of unknown quality.

Pasteurization is too important a public health measure to leave to individual caprice. The process should be under official supervision. Further, pasteurized milk should be labeled as such or simply "heated milk," stating the degree of heat and the length of time, and the date on which the process was done.

Pasteurization is sometimes objected to because it does not destroy heat-resisting toxines which are supposed to be in milk. The occur-

rence of such poisons is a mere assumption. Even if they exist in milk they would be in the raw milk as well as in the heated milk. The true exotoxins are all killed at 60° C. for 20 minutes.

Fig. 71.—Automatic Temperature Recorder for Pasteurizers.

Theoretically the best place to pasteurize milk is in the home. Practically the best place is at some central station, where it may be done scientifically under official surveillance.

Methods of Pasteurization.—There are three well-known methods by which milk may be pasteurized: (1) the flash method; (2) the holding method; (3) in the bottle.

The *flash method* consists of heating the milk momentarily to a temperature of about 178° F. and chilling it at once. This method is sometimes incorrectly called commercial pasteurization. It does not give uniform results, is not entirely reliable, and does not meet with the approval of the sanitarian. The method, however, is rapid, cheap, and is much in vogue.

The *holding method* consists in heating the milk to the desired temperature, say 65° C., and then holding it in a suitable tank or series of tanks at that temperature for a given period of time, say 30 or 45 minutes. This method has proven satisfactory in practice under commercial conditions.

Pasteurization *in the bottle* is the perfection of the art. It is the ideal method, because the danger, however slight, of recontamination is entirely eliminated. In order to pasteurize milk in bottles the bottles must be well sealed with a crown cork and cap, or equally effective stopper. The bottles containing the milk may either be immersed in a water bath, brought to the proper temperature, held there a sufficient length of time, and then chilled; or the methods used in beer pasteurization, such as the Loew pasteurizers, may be used. In this case the bottles are subjected to a spray or shower of heated water.

Freeman's pasteurizer for heating milk in individual feeding bottles

in the home is most serviceable. The modification of Mr. Nathan Straus is shown in Fig. 72. It is used as follows:

After the bottles have been thoroughly cleaned they are placed in the tray (A) and filled with the milk or mixture used for one feeding. Then put on the corks or patented stoppers without fastening them tightly.

The pot (B) is now placed on the wooden surface of the table or floor and filled to the supports (C) with boiling water.

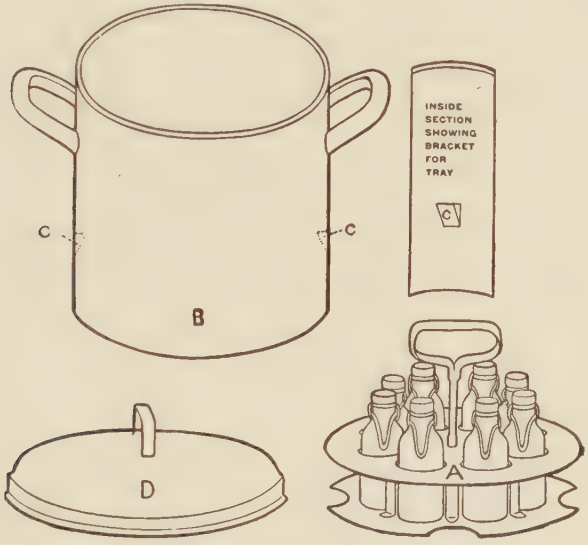


Fig. 72.—Straus Home Pasteurizer.

Place the tray (A) with filled bottles into the pot (B) so that the bottom of the tray rests on the supports (C), and put cover (D) on quickly.

After the bottles have been warmed up by the steam for five minutes, remove the cover quickly, turn the tray so that it drops into the water, replace the cover immediately. This manipulation is to be made as rapidly as possible to avoid loss of heat. Thus it remains for twenty-five minutes.

Now take the tray out of the water and fasten the corks or stoppers air-tight. Cool the bottles with cold water and ice as quickly as possible, and keep them at this low temperature until cold.

Use the milk from the bottles and do not pour it into another vessel.

The milk should not be used for children later than twenty-four hours after pasteurization.

Emphasis is laid on the fact that only fresh, clean milk, which has been kept cold, should be used.

The Effect of Heat upon Milk.—The changes produced in milk by heating depend upon the degree of heat and the length of exposure. Milk heated to 60° C. for a short time does not appreciably affect its chemical and physical properties. The boiling of milk, however, produces pronounced changes. In the main, these consist of a partial decomposition of the proteins and other complex nitrogenous derivatives; diminution of the organic phosphorus and an increase of inorganic phosphorus; precipitation of the calcium and magnesium salts and the greater part of the phosphates; expulsion of the greater part of the carbon dioxid; caramelization or burning of a certain portion of the milk sugar, causing the brownish color; partial disarrangement of the normal emulsion, and coalescence of some of the fat globules; coagulation of the serum albumin, which begins at 75° C.; the ferments are killed.

Boiled milk has a cooked taste which appears at about 70° C. This is due perhaps to the decomposition of certain of the proteins in the milk. The loss of certain gases also alters the taste, so that milk heated in closed vessels has a less pronounced flavor than if heated in open vessels.

Milk heated in the open air forms a pellicle which renews if it is removed. This scum forms when milk reaches about 60° C. It consists of:

Fatty matter	45.42 per cent.
Casein and albuminoid.....	50.86 per cent.
Ash	3.72 per cent.

Milk heated in closed vessels does not form a pellicle, even when the temperature reaches the boiling point. It seems that this pellicle is due mainly to the drying of the upper layer of the liquid.

After milk has been heated to 65° C. or over for half an hour, the cream does not rise well, if at all, owing to the increase in the viscosity of the fluid in which it is emulsified. The clusters of fat droplets which are agglutinated into masses in normal milk are broken down by heating, and the globules are more homogeneously distributed throughout the fluid.

It has been observed that cooked milk coagulates with rennin more slowly than raw milk. This effect is noted often at temperatures of 80° to 90° C., but has not been observed in milk heated to 60° C. for 20 minutes. The curd produced by rennin coagulation in cooked milk is softer, less tough, and more flocculent than that produced by rennin coagulation in raw milk. This is believed to be an advantage favoring the digestibility of heated milk. Cooked milk is said to be constipating. This is explained by the fact that cooked milk contains comparatively few bacteria and is, therefore, less irritating than raw milk.

THE BACTERIOLOGICAL EXAMINATION OF MILK

The Number of Bacteria.—No known method can give an enumeration of all the bacteria in milk. Some are aerobes, others anaerobes; some require alkaline, others acid media; some grow best at room temperature, others only at blood temperature; and some grow slowly or not at all upon ordinary media. The methods in use, therefore, are those which have been shown by experiments to give the highest counts and the maximum information under ordinary conditions.

For the sake of uniformity methods should follow the report of the Committee on Standard Methods of Bacterial Milk Analysis of the American Public Health Association.[1]

The samples must be collected and kept in such a manner as to prevent either any addition of bacteria from without or multiplication of the bacteria originally present. Whenever possible, and especially in the selection of certified milk samples, an original package should be taken, placed in a suitably iced case, and brought at once to the laboratory. Samples of market milk may be collected in the same manner as water samples, in sterile, wide-mouthed, glass-stoppered four-ounce bottles. Care should be taken to secure a sample which is thoroughly representative of the milk to be examined. This may be done by pouring the milk back and forth into a sterile receptacle, or shaking the milk thoroughly with the receptable turned upside down. In taking samples from tanks it is allowable to stir thoroughly with a long-handled dipper. Generally speaking, the shorter the time between collection and examination of milk samples the more accurate will be the results. For routine work the attempt should be made to plate within four hours of the time of collection. Too much stress cannot be laid on the importance of keeping the samples properly iced during this interval. They should be kept below 40° F., but care should be taken that they are not frozen.

The standard medium for routine enumeration of bacteria in milk is: agar, 1 per cent., reaction +1.5, Fuller's scale.[2] Milk should always be diluted before plating, for the reason that whole milk produces a turbidity of the agar, and because the bacteria cannot well be dispersed without diluting, and the resulting colonies are so close that they interfere with each other. The milk is diluted in the proportion of 1-10, 1-100, 1-1,000, 1-10,000, 1-100,000, or 1-1,000,000. For certified milk 1-100 dilution should be used. Ordinary potable water, sterilized, may be used for dilutions. The number of bacteria present may be estimated approximately before dilutions are made by direct micro-

[1] *American Journal of Public Hygiene*, August, 1910, VI, 3, p. 315.

[2] Æsculin bile salt, agar, lactose, litmus agar, and whey agar may also be used.

scopic examination of a properly prepared sediment. Otherwise it is necessary to make a range of dilutions therefrom, selecting for record the count obtained on that plate which yields between 40 and 200 colonies. A plate containing more or less than these numbers will not give reliable results. Porous, earthenware Petri dish covers are recommended as superior to glass, since they absorb the excess of moisture and thus help prevent spreaders. Another method of preventing spreaders is to invert the dishes and place in the glass cover of each a strip of sterile filter paper moistened with one large drop of glycerin.

The plating should always be checked by duplicate controls, and a blank plate should be made with each series for control of the sterility of the agar, water, air, Petri dishes, pipettes, and methods. The plates should be incubated at 37° C. for 48 hours, or may be grown at 21° C. for five days. Only those colonies should be counted which are visible to the naked eye or may be seen readily by a low power lens. The result should always be expressed in round numbers. It is misleading to state that a milk contains 2,140,672 bacteria per c. c. This gives a false and exaggerated notion of the accuracy of the method. At best the results are only an average approximation. Results should be expressed in accordance with the recommendations of the Commission on Standards of the New York Milk Committee.[1]

The Kinds of Bacteria.—We still lack satisfactory routine methods for determining the kinds of bacteria found in milk. If the plates are made with gelatin it will give the relative proportion of liquefiers. By the use of Endo's medium or lactose litmus agar the number of acid-producing bacteria may be determined. The number of fermenting organisms may be estimated by planting progressively smaller quantities in fermentation tubes containing glucose or other sugar; or by the use of the Wisconsin curd test. The presence of gas-producing organisms in abundance usually indicates dirty conditions of stables, cows, or containers.

To determine the number of proteolytic bacteria in milk place 1 c. c. of sterile skim milk into a Petri dish, then add the proper dilution of milk in question, and finally pour in molten sugar-free agar. Incubate 48 hours, and then wash the surface with a dilute solution of acetic acid. Count the number of colonies surrounded by a clear zone, which is taken to represent proteolysis or breaking down of the protein.[2]

Typhoid bacilli may be isolated on Endo's medium, and diphtheria upon Loeffler's blood serum. Other pathogens require special technique applicable to each case. The number of streptococci in milk may be estimated by the direct examination of stained smears. The chains are more readily counted if the milk is first incubated at 37° C. for

[1] *Public Health Reports*, Vol. XXVII, 19, May 10, 1912.
[2] Hastings: *Cent. f. Bakt. u. Parasitenk.*, Abt. II, Bd. X, p. 384.

6 or 8 hours. In the estimation of streptococci only the longer chains are considered. The presence of streptococci and an approximation as to their number may also be determined by planting the milk upon the surface of blood agar and studying the fine dewdrop-like colonies.

A few streptococci will be found in most sediments from milk. They are seldom found to any great extent by direct microscopic examination of clean milk. Occasionally a sample will be found crowded with long chains. More often streptococci, if present, are in the form of diplococci or very short chains. The common interpretation is to regard the short chain varieties as probably harmless, while long chains are regarded as more apt to indicate inflammatory reactions. A milk containing these in large numbers may not be a safe article of diet.

Ruediger points out that *Streptococcus lacticus* can be differentiated from *Streptococcus pyogenes* by means of blood agar plates. *Streptococcus pyogenes* produces small colonies surrounded by a large zone of hemolysis, whereas *Streptococcus lacticus* produces green or grayish colonies with very little or no hemolysis.

Streptococcus lacticus has no sanitary significance, as it is found in nearly all samples of clean, soured, or fresh milk, and very often in the healthy milk ducts. *Streptococcus pyogenes,* on the other hand, seems to occur but rarely in milk, and is indicative of the existence of an inflamed condition of the udder of the cow furnishing the milk.

The presence of *Bacillus ærogenes capsulatus,* the gas bacillus of Welch, may be determined by heating some of the milk to 80° C. for one hour and then incubating the sample at 37° C. If the sample contains this microorganism it will show active fermentation with gas production within 24 hours (sometimes as soon as 6 hours), with the development of an odor of butyric acid.

The demonstration of tubercle bacilli in milk depends upon animal experimentation. Guinea pigs are injected subcutaneously with 5 c. c. of sediment obtained by centrifuging, or with cream, or both. The guinea pigs that do not die in two months are tested with sufficient tuberculin (O. T.) to cause the death of the tuberculous animals in 24 hours. Two c. c. of the crude tuberculin is injected subcutaneously for this purpose.

MICROSCOPIC EXAMINATION

There are three methods of making a microscopic examination of milk in current use.

(1) The Stewart-Slack Method.—Two c. c. of milk are placed in a glass tube closed at both ends with a rubber stopper. This is centrifuged for 10 minutes at a speed of from 2,000 to 3,000 revolutions per minute. The sediment upon the rubber stopper of the distal end

of the tube is mixed with a drop or two of water and spread upon a slide in a thin even layer, covering a space of about four square centimeters. This is dried and stained with methylene blue. The microscopic examination reveals the character of the milk as judged from the approximate number of pus cells and presence of streptococci in long chains. It has been found that the number of cocci, bacilli, or chains in the 1/12 oil immersion field, multiplied by 10,000, gives a rough approximation of the number of bacteria in a cubic centimeter of the whole milk.

The results of this method vary considerably with details of individual manipulation, with the speed of the centrifugal machine, with the time allowed for centrifugation, and other factors.

(2) The Doane-Buckley Method.—In this method the number of leukocytes are counted in the chamber of the Zeiss blood counter, which contains just 0.0001 c. c. Ten c. c. of milk is centrifuged at 2,000 revolutions per minute for four minutes. The fat is removed with a cotton swab and again centrifuged for one minute. The fat is again carefully removed, for any appreciable amount of fat will interfere with the counting. The supernatant fluid is now pipetted off and two drops of a saturated alcoholic solution of methylene blue are added to the sediment, which is thoroughly mixed and warmed in boiling water for two or three minutes, which favors the staining of the cells. The sediment is now diluted to the 1 c. c. mark with water. Some of this is transferred to the counting chamber and the number of cells counted with a dry lens. The number of cells in the counting chamber multiplied by 1,000 gives the number per c. c. in the milk.

(3) The Prescott-Breed Method.—A capillary tube is prepared, arranged to receive a rubber bulb at one end, and marked carefully to deliver 0.01 c. c. After a most thorough mixing of the milk, 0.01 c. c. is removed with the sterilized pipette and spread uniformly over a square centimeter on an ordinary microscopic slide. It is allowed to dry and is fixed with methyl alcohol, after which the fat is dissolved from it by the use of xylol. The smear is then stained either with methylene blue or preferably with one of the blood stains, the Jenner stain or Wright stain being useful for this purpose. If the staining is so deep as to make the specimen too opaque for proper study, it is slightly decolorized with alcohol, which removes the stain from the general sediment more readily than it does from the bacteria or the tissue cells. The stained smear is studied under a twelve-inch immersion lens. The draw tube is adjusted so that the field of the microscope covers exactly 15 millimeters, and under these circumstances the number of bacteria present in the 0.01 c. c. is exactly 5,000 times the number found in a microscopic field. The counting of a large number of fields (100 fields) and averaging the results multiplied by this number will, there-

fore, give approximately the number of cells or bacteria contained in 0.01 c. c. of milk.

CHEMICAL ANALYSIS OF MILK

Total Solids.—The total solids in milk consist chiefly of the fats, sugar, proteins, and inorganic salts. The United States standard requires 12 per cent. of the milk to consist of total solids, 8.5 per cent. of which shall be solids, not fat, and 3.25 per cent. fat. In some states the requirement for total solids is as high as 13 per cent., in others 11.5 per cent.

Determination of Total Solids.—The total solids may be determined either by:

(1) The use of Richmond's slide rule.
(2) The Babcock asbestos method.
(3) By evaporation and direct weighing.

Richmond's Slide Rule.—This is a device by which the total solids may be determined fairly accurately by the use of the formula of Hehner and Richmond. It is necessary to know the correct specific gravity and the amount of fat. From this the total solids is determined by the following formula:

$$\text{T S}=(\frac{\text{G}}{4})+1.2\ \text{F}+.14$$

in which T S equals total solids, G the last two units of the specific gravity and any decimal. Thus, if the specific gravity is 1.0295, G= 29.5. F represents the percentage of fat. In using the slide rule the operation is conducted in two stages. First, the lactometer reading is corrected for temperature. The observed lactometer reading is brought opposite the 60° and the correct lactometer reading read off opposite the observed temperature. Second, the arrow of the slide is set opposite the observed percentage of fat, and the total solids are read off opposite the corrected specific gravity reading on the scale marked "specific gravity." The results obtained by the use of Richmond's slide rule agree quite closely with those obtained by direct weighing.

This formula may also be used to determine the percentage of fat provided the specific gravity and total solids are known.

The Babcock Asbestos Method.—The milk is placed upon a filter paper cartridge filled loosely with freshly ignited woolly asbestos, subjected to a temperature of 100° C. until weight is constant, and then cooled and weighed. The gain in weight represents the total solids of the amount of milk taken. The advantage in this method is that the cartridge may then be slipped into the Soxhlet extraction apparatus and used for the determination of fat.

Weighing.—About 5 c. c. of milk are weighed in a tared platinum dish, evaporated exactly two hours on a steam bath, the outside wiped

dry, and then cooled to constant weight in a desiccator. The weight of the residue represents the total solids of the milk.

Determination of Ash.—The platinum dish containing the total solid residue is carefully heated in the flame, avoiding spattering and heating above a dull red glow. When the residue has become white, or nearly so, it is cooled in a desiccator and again weighed; the difference between the final weight and the original weight of the empty dish represents the amount of mineral matter in the amount of milk taken. The ash is saved for the tests for boron compounds, carbonates, and other non-volatile mineral preservatives.

Determination of Fats.—The determination of the quantity of butter fat contained in milk is of considerable economic importance and is included as a routine in all milk laboratories. There are several methods by which the fat in milk may be accurately determined.

(1) Babcock Method.—The Babcock method is the most convenient and is sufficiently accurate for ordinary purposes. It cannot be carried out without considerable special apparatus, including a centrifuge, special graduated flasks and pipettes. The principle of this method depends upon separating the fat by means of the addition of sulphuric acid. The mixture is centrifugalized so that the fat rises into the neck of the specially graduated flask, and the percentage may be read off directly. The method is carried out as follows:

In the special graduated flask are mixed:

17.5 c. c. milk.
17.5 c. c. of sulphuric acid (specific gravity 1.82-1.83).
2 c. c. amyl alcohol (optional).

The acid must be run slowly down the side of the flask under the milk and the whole mixed, without splashing, by imparting a rotary motion to the contents of the bottle. The mixture is centrifugalized for 4 minutes; boiling water is then added until the liquid rises to the bottom of the neck of the flask, and the centrifugalization is repeated for 2 minutes. Again add boiling water until the top of the column is near but safely under the top of the scale, and centrifugalize a third time for 1 minute. By this time the fat in the neck of the bottle should be clear, yellow, and liquid. The length of the column of fat is considered as extending from the bottom of the line of contact with the liquid below to the top of the meniscus above. The length of the column of fat is measured by means of a pair of dividers, which are first adjusted to the length of the column of fat, and the percentage read by touching one point of the dividers to the zero mark on the scale, when the upper point will indicate the percentage of fat in the milk. The mixing of sulphuric acid with the milk generates consid-

erable heat, which should be maintained, so that at the time of taking the reading the contents of the bottle register between 55°-60° C. Care should be taken to use none but authoritatively tested and guaranteed bottles.

(2) The Werner-Schmidt Method.—This method is slower than the Babcock, especially when many samples are to be analyzed, but it can be done with improvised apparatus and readily procurable materials. Ten c. c. of milk are added to 10 c. c. of concentrated hydrochloric acid in a 50-c. c. test tube, shaken, and boiled until dark brown in color. The mixture is then cooled in water and 30 c. c. of washed ether added, the stopper inserted, and thoroughly agitated. When the two layers have separated the upper layer containing the ether and dissolved fat may be withdrawn by means of a pipette, or blown out with the assistance of a double tube, such as is used in wash-bottles, the delivery tube extending into the ether layer almost to the line of demarcation between the ether and the acid-milk mixture. The ether containing the extracted fat is transferred to a weighed flask. The extraction is repeated with several fresh, smaller portions of ether (about 10 c. c.), and the whole of the ether used is collected in the weighed flask. The ether is then distilled off or permitted to evaporate at a low temperature. The residuum of fat is heated to constant weight in an air bath, cooled, and weighed. Since the milk is measured and not weighed, a correction must be made accordingly.

Example.—Amount of milk used equals 10 c. c. Specific gravity of sample equals 1.029. Weight of milk used, therefore, equals 1.029×10, which equals 10.29 grams. The weight of the fat found equals 0.386 gram. Percentage of the fat in the original milk is determined from the following equation:

$$10.29 : 0.386 :: 100 : x$$

$x = 3.97$, or the percentage of fat in the original milk.

(3) The Soxhlet Extraction Method.—This is the most accurate method for determining fats in milk and other substances. The principle depends upon the complete extraction of all the fat by continuous washing with ether. The only error in this method consists in the fact that substances other than fats are soluble in ether and are included in the weight. This error in milk is negligible. The process requires a coil of thick filter paper free from substances soluble in ether and alcohol, and a Soxhlet extraction apparatus. Instead of the coil of filter paper a specially prepared cartridge of filter paper, which fits loosely within the cylinder of the Soxhlet apparatus, may be used. When the cartridge is used it is best to plug its open end with absorbent cotton, in order to prevent the escape of fine particles of the contained substance.

A definite weight of milk, about 5 grams, is applied to the coil of filter paper or cartridge, in one of two ways. A small beaker containing the required amount is weighed and the coil is placed into it and kept there until nearly the whole has been absorbed. The coil is then carefully withdrawn and placed, dry edge downward, upon a sheet of glass. The beaker is then weighed again, and the loss in weight, which represents the amount of milk absorbed, is noted. Another method is to weigh the beaker containing the milk and a small pipette. The necessary amount of milk is then transferred to the coil with the pipette, after which the weight of the beaker and pipette containing the remaining milk is noted. The difference represents the weight of the milk absorbed. The coil or cartridge is then dried in an air bath at 100° C. for an hour or more, when it is ready for insertion into the extractor.

The three separate parts of the Soxhlet extraction apparatus, consisting of the flask, the cylinder, and the condenser, are joined together and mounted upon a water bath or an electrically heated plate. Before the operation is begun the exact weight of the flask must be determined. The ether is then added, and as it volatilizes the vapor passes upward through the side tube into the extractor, and thence to the condenser, where it falls upon the substance to be extracted. As the process continues the condensed liquid accumulates in the cylinder and gradually rises until it reaches the bend of the siphon in the cylinder part of the apparatus. When full the siphon acts and discharges back into the flask, until the entire liquid is returned to its starting point. During its accumulation in the cylinder it dissolves the fats or other ether soluble substances which are carried in solution into the flask. The process is continued until this siphoning action repeats itself again and again as long as is necessary, so that the whole of the extracted matter is finally within the flask. The fat, being non-volatile, remains in the flask while the ether is revolatilized and sent continually on its errand. On the completion of the process the ether is permitted to collect in the cylinder, but before it reaches the level of the siphon the flask is disjoined. The remaining ether is expelled cautiously and the flask with its content is placed in an air bath maintained at 100° C. and dried to constant weight. The increase in the weight of the flask represents the amount of matter extracted.

Example.—The weight of milk absorbed by the filter paper was 5.160 grams. The increase in the weight of the flask was 0.161 gram. The amount of fat present in the sample is then obtained by the following equation:

$$5.16 : 0.161 :: 100 : x$$

$x = 3.20$, or the percentage of fat in the milk.

Determination of Milk Sugar.—The amount of lactose in milk may be determined chemically by the reduction of copper sulphate in Fehling solution, or optically by means of the polariscope.

(1) Method by Fehling's Solution.—To 25 grams of milk add 0.5 c. c. of 30 per cent. acetic acid; shake; let stand 3 minutes; then add 100 c. c. of boiling water; again shake; add 25 c. c. of alumina cream; again shake, and let stand for 10 minutes; filter through a wet pleated paper filter and wash the residue until the washings and filtrate total 250 c. c., representing a dilution of 1-10 of the original milk; this dilutes the sugar content of the liquid to somewhat less than 0.5 per cent. This is then titrated with Fehling's solution in the usual manner, namely: fill a burette with sugar-containing liquid, place 10 c. c. of Fehling's solution (representing 0.067 gram of milk sugar) in a flask, and heat to boiling. Run in the liquid from the burette in small portions, maintaining the contents of the flask at boiling point until the liquid in the flask loses its original blue color, which marks the end point of the reaction.

Fehling's solution is made up in two solutions: 1. Dissolve 34.639 grams of pure sulphate of copper in distilled water and dilute it to a liter. 2. Dissolve 173 grams of potassium sodium tartrate (Rochelle salt) in distilled water, add 100 c. c. of sodium hydrate solution of 1.393 specific gravity, and dilute the mixture with distilled water to a liter. Equal parts of solution 1 and 2 are mixed in a boiling-flask of about 300 c. c. capacity. The amount of copper contained in 10 c. c. of solution 1 requires for its reduction 0.050 gram of dextrose, or 0.667 gram of lactose.

Polariscope Method.—The polariscope, the quantities used, and the factors employed in the polariscope method vary with different types of instruments. Perhaps the most satisfactory is the Schmidt and Haenzsch half-shadow type. This possesses the advantage of doing away with the matching of colors, and hence may be used by those who are color-blind, and even with those having normal color vision it gives the most satisfactory results.

To 70.65 grams of milk add an excess (3 c. c.) of an acid nitrate of mercury solution and mix thoroughly by shaking. The acid nitrate of mercury solution is made by adding one part of weight of mercury to two parts of nitric acid, S. G. 1.42, and, after the reaction has ceased, adding an equal volume of distilled water. The object of adding the acid nitrate of mercury to the milk is to remove the albumin and fat in the form of a curd, leaving the sugar as the only optically active constituent of the clear serum. The milk containing the acid is now diluted to 102.5 c. c. with distilled water and again thoroughly mixed. Filter through a dry pleated filter and take the polarimeter reading without delay in a 200-mm. tube. When an excess of acid nitrate of

mercury is added to the sugar-containing liquid the latter quickly begins to decompose, with the evolution of gas; on the other hand, an excess must be present in order to obtain a clear, easily filtered liquid.

The percentage of lactose is the product of the factor 0.0209 (this factor is applicable to these conditions only) multiplied by the number of minutes of dextrorotation. The definite directions for this particular kind of work do not accompany the instrument used. The factor should be determined or confirmed by comparing with lactose solution of known strength. Some polarimeters are graduated directly in sugar percentages instead of degrees and minutes, in which case care must be taken that the graduations correspond to the particular form of sugar under investigation, or, if not, that a suitable correction is made.

Determination of Proteins.—It is not usual to estimate the proteins in a sanitary analysis of milk, since different specimens of milk vary very little in this regard, and since there is little inducement for sophistication, as far as the proteins are concerned.

(1) Method by Difference.—If we know the weight of total solids in milk and subtract therefrom the weight of the fat, ash, and sugar, the difference will represent the proteins. This method is sufficient for ordinary purposes. To estimate the nature of the various proteins requires special skill in organic analysis.

(2) Kjeldahl Method.—The milk is mixed with sulphuric acid and digested in a flask until it is completely charred and becomes clear again. The residue will then contain all of the nitrogen in the form of ammonium sulphate, which is determined in the usual way. The total nitrogen multiplied by the factor 6.38 gives the total protein. The method is carried out as follows:

Gunning Modification.—An accurately weighed amount (about 5 grams) of milk is placed in a 500-c. c. Kjeldahl digestion flask and digested with 10 grams of potassium sulphate and 15 c. c. of concentrated nitrogen-free sulphuric acid. The digestion is carried out over a free flame, using care to heat gradually at first; the process is considered complete when the liquid becomes clear (about 2 hours). The contents of the flask are cooled and 200 c. c. of water and sufficient saturated sodium hydroxid solution to neutralize the acid and to make the solution strongly alkaline are added. The nitrogen, which has been converted into ammonium sulphate, is now distilled through a block tin tube into a definite amount of standard acid, and the acid titrated back with standard alkali, using cochineal or alizarin as indicator. The amount of nitrogen can be calculated from the results. Total nitrogen multiplied by 6.38 gives total protein.

Water.—Milk is still frequently sophisticated by the addition of water. A watered milk may be suspected from a low specific gravity,

or may be detected unerringly by the index of refraction of the milk serum.

Refractometer Reading.—This test depends upon the fact that the salts dissolved in undiluted milk in the concentration in which they exist in the milk serum, as prepared under standard conditions, give a reading of not less than 39 upon the scale of a Zeiss refractometer at a temperature of 17.5° C. Distilled water gives with the same instrument a reading of 15. Milk reading below 39 is certainly watered.

Refractometer reading is obtained as follows:

The milk serum is prepared by adding 2 c. c. of a 25 per cent. acetic acid (S. G. 1.035) to 199 c. c. of milk at about 20° C. and mixing well. Heat the mixture to 70° C. Maintain this temperature for 20 minutes. Cool quickly to room temperature by means of cold water, and filter until nearly or quite clear. Do not discard the curd, as it can be used to test for the presence of artificial colors. The refractometer reading is taken with the filtrate at 17.5° C., this temperature being maintained by means of a large body of water at the same temperature surrounding the milk container.

If a refractometer is not at hand practically the same information can be obtained from the milk serum by taking its specific gravity with a Westphal balance or a pycnometer.

Reaction.—The acidity of milk is determined by titration with a solution of sodium hydroxid, using phenolphthalein as the indicator.

Take 50 c. c. of milk and add a few drops of alcoholic phenolphthalein solution. From a burette run in 0.1 normal sodium hydroxid solution with constant stirring until the pink color in the milk persists about 15 seconds. The carbon dioxid in the atmosphere fades out the phenolphthalein color by converting the sodium hydroxid into sodium bicarbonate, hence the determination must be made rapidly, and a rather faint but not very permanent pink color marks the end point.

The acidity of milk is usually expressed in terms of lactic acid, although when fresh it is caused by other organic acids. To convert the amount of $\frac{N}{10}$ sodium hydroxid solution necessary to neutralize the acidity in 50 c. c. of milk into percentages of lactic acid, multiply the number of cubic centimeters of $\frac{N}{10}$ NaOH by 0.018.

The results of these titrations are recorded in three different ways: (1) In this country the calculations are reduced to terms of lactic acid. Thus, 1 c. c. of $\frac{N}{1}$ NaOH neutralizes 0.02 gram of lactic acid; (2) in degrees of acidity, by which is meant the number of cubic centimeters of $\frac{N}{10}$ NaOH required to neutralize 100 c. c. of milk; (3) in

German degrees of acidity, meaning the number of cubic centimeters of $\frac{N}{4}$ NaOH per 100 c. c. of milk. For transposition purposes the following equivalents are given:

1 degree (U. S.) of acidity.....0.009 per cent. lactic acid
1 degree (German) of acidity....0.0225 per cent. lactic acid
1 degree (German) of acidity....2.5° (U. S.) acidity

Rühm[1] has recommended the following test for detection of beginning acidification in mixed milks of two or more cows: Ten c. c. of 68 per cent. alcohol is added to 10 c. c. of the milk to be tested. If there is immediate coagulation the acidity is above 8°. More advanced acidity may be detected by boiling a small amount of milk for a few moments in a test tube. Coagulation appears if the acidity is above 10°. These are convenient tests that may be applied at the dairy.

Milk has a variable acidity when it coagulates; that is, when it throws its caseinogen out of solution. Milk containing about 0.225 per cent. of acid will coagulate upon heating. This may be prevented by first neutralizing with an alkali, such as sodium carbonate. The amount of acidity in a particular sample of milk is no safe criterion as to whether it will coagulate or not during pasteurization. This can only be determined with certainty by first testing a small portion.

Specific Gravity.—The specific gravity of milk is taken either (1) with the lactodensimeter, (2) with the Westphal balance, or (3) upon an ordinary chemical balance, with a pycnometer.

The Quevenne lactodensimeter is recommended for the determination of the specific gravity. It is made like an ordinary aerometer and divided into degrees which correspond to a specific gravity from 1.014 to 1.040, or only from 1.022 to 1.038, since by the latter division a greater space is gained between the different degrees without unduly lengthening the instrument. From such a lactodensimeter one can easily read off four decimal places.

The milk, the specific gravity of which is to be determined, is well shaken and poured into a high-class cylinder of suitable diameter; the lactodensimeter is dropped in slowly, in order to prevent its bobbing up and down. (The bulb should be free from adhering air bubbles.) The figures on the stem are the second and third decimals of the numbers of the specific gravity, so that 34 is to be read 1.034. For this examination the temperature of the milk must be 15° C. (60° F.); if it is not, the specific gravity of the milk at 15° C. must be calculated from the specific gravity found and from the temperature, for in milk inspection and analysis this is the standard.

[1] Rühm: *Zeitschr. f. Fleisch u. Milch-hyg.*, Vol. XX, 1910.

Westphal Balance.—This instrument is more accurate than the lactometer. It is in equilibrium when the sum of the weights equals the specific gravity of the liquid.

Taking the specific gravity of the whole milk does not of itself detect either watering or skimming, since, if these practices are done artfully, the specific gravity of the milk may remain unaltered. The specific gravity of normal milk serum is about 1.0287.

Heated Milk.—Milk that has been heated above 79° or 80° C. may be detected by the fact that the enzymes are killed. Several methods are used; the most convenient, perhaps, is Dupouy's method. A few drops of a freshly prepared solution of diamidobenzin in water (1-4) and a little hydrogen dioxid are added to 5 c. c. of milk. With raw milk a coloration appears, while with milk that has been heated to 79° C. or over no color is produced. Other tests, such as the Storch method or Arnold's guaiac method, are described below under "Tests for Enzymes." A test for heated milk has recently been devised by Frost and Ravenel, who find a difference between the staining of the cells when subjected to an aqueous solution of safranin.

Tests for Enzymes, and Their Significance.—The following tests are those most frequently used:

Catalase Test.—Ten c. c. of the milk to be tested is mixed with 10 c. c. of a 3 per cent. (by volume) hydrogen peroxid. The mixture is placed in a Lobeck tube and the stopper tightly inserted. Then the tube for measuring the liberated oxygen is filled with water and inserted into the perforated stopper, pushing out the small hard rubber button. The mixture of milk and hydrogen peroxid is immersed up to the stopper in a water bath at 37° C. and left there for two hours. The oxygen that is liberated replaces the water in the graduated tube on which the readings are made. Larger quantities of milk (15 c. c.) and less hydrogen peroxid (3 c. c.) give more satisfactory readings for pasteurized milk.

According to Auzinger, the liberation of much gas by this test occurs (1) with physiologically changed milk, as is the case with colostrum and with milk from old milkers; (2) in the case of pathologically changed milk, as in mastitis and other febrile diseases; or (3) in milk containing a large number of bacteria. The test for catalase, therefore, is of assistance in detecting old, bacteria-laden, or abnormal milk.

Reductase Test.—(1) *Schmidt-Muller or Slow Reductase Test.*—The reagent is made by adding 195 c. c. of distilled water to 5 c. c. of a saturated alcoholic solution of methylene blue (zinc chlorid double salt). This reagent should be boiled every day before using. The test is made by adding to 20 c. c. of milk in a test tube 1 c. c. of the reagent, mixing, sealing with melted paraffin, and then incubating at

45° C. in a water bath. According to Rühm,[1] fresh milk remains blue for 12 hours or more, and "infected" milk decolorizes in less than one hour. Reductases, according to Rühm, are increased by acid-forming bacteria, but not by alkaline producers. Auzinger,[2] who uses 0.5 c. c. of the reagent in 20 c. c. of milk, states that, on holding the mixture at 38° to 40° C., milk not decolorizing in seven hours contains less than 100,000 bacteria per c. c.; that which decolorizes in 2 to 7 hours contains 100,000 to 300,000; and that which decolorizes in ¼ to 2 hours contains 300,000 to 20,000,000 bacteria per c. c.

(2) *Schardinger or Hastened Reductase Test.*—The reagent is made by adding 190 c. c. of distilled water and 5 c. c. of formaldehyde solution to 5 c. c. of saturated alcoholic solution of methylene blue (zinc chlorid double salt).

The test is made by adding to 10 c. c. of milk 2 c. c. of the reagent in a test tube, mixing well, sealing with melted paraffin, and holding at 37° C. in a water bath. By the test, according to Auzinger,[2] good milk reduces the color in 8 to 12 minutes, milk rich in bacteria reduces in 5 minutes or less, and when colostrum is present two or more hours are required.

Of the two reductase tests, according to Schardinger,[3] reduction by the slow method is due to ferments produced by bacteria, while by the hastened method reduction is due to the natural ferments of milk.

The slow reductase test is of assistance in detecting old milk, and the hastened reductase test offers a convenient and reliable method for detecting and testing the efficiency of pasteurization.

Storch Test.—This test is made by adding to 5 c. c. of milk one drop of 0.2 per cent. H_2O_2 and two drops of a 2 per cent. solution of paraphenylendiamin and thoroughly mixing. The reagent must be freshly made at least every two weeks.

Wilkinson and Peters Test.[4]—This test is made by adding to 10 c. c. of milk 2 c. c. of a 4 per cent. alcoholic benzidin solution and two or three drops of acetic acid, then mixing well, and adding 2 c. c. of 3 per cent. H_2O_2.

Guaiac Test.[5]—The reagent is made by adding one part of guaiac to ten parts of acetone. To make the test several drops of 0.2 per cent. H_2O_2 and 1 c. c. of the guaiac solution are added to 10 c. c. of milk. The reaction appears in one to three minutes.

Bellei Test.—The test is made by adding to 10 c. c. of milk 3 drops of 1.5 per cent. aqueous solution of ortol and two drops of 3 per cent. H_2O_2.

[1] Rühm: *Zeit. f. Fl. u. Milch-hyg.*, Vol. XX, 1910.
[2] Auzinger: *Ibid.*, Vol. XX, 1910.
[3] Schradinger: *Arch. f. Kinderheilk.*, Bd. 58, H. 5-6.
[4] Wilkinson and Peters: *Zeit. Nahr. u. Genuszm.*, 1908, p. 172.
[5] Arnold and Mentzel: *Milch Zeit.*, 1902, p. 31.

The Storch, Wilkinson and Peters, guaiac, and Bellei tests are used primarily to detect heating above 70° C., and are of little practical value, as heating to such high temperatures is seldom resorted to in this country.

REFERENCES

Sommerfeld, Paul: "Handbuch der Milchkunde," J. F. Bergmann, Wiesbaden, 1909.

Swithinbank, Harold, and Newman, George: "Bacteriology of Milk," E. P. Dutton & Co., 1903.

"Milk and Its Relation to the Public Health," *Hyg. Lab. Bull. No. 56*, U. S. P. H. & M. H. S. Various authors.

Rosenau, M. J.: "The Milk Question," Houghton, Mifflin & Co., 1912.

Savage, Wm. G.: "Milk and the Public Health," Macmillan & Co., Ltd., London, 1912.

Report of the Commission on Milk Standards, N. Y. Milk Committee, *Public Health Reports,* U. S. P. H. & M. H. S., xxvii, 19, May 10, 1912.

Barthel, Chr.: "Die Methoden zur Untersuchung von Milch und Molkereiprodukten." Leipzig, 1911. English edition translated by Goodwin.

Farrington and Woll: "Testing Milk and Its Products," 21st edition, Mendata Book Co., Madison, Wis., 1912.

CHAPTER III

ANIMAL FOODS: MEAT, FISH, EGGS, ETC.

MEAT

The universal consumption of meat as a daily article of diet by civilized man is of more recent origin than is generally supposed. McCulloch[1] states that "so late as 1763 the slaughter of bullocks for the supply of the public markets was a thing wholly unknown, even in Glasgow, though the city then had a population of 30,000." In the past decade or two the consumption of meat has increased enormously, especially in the United States and England, owing to the development of cheap refrigerator processes, canning, and increased facilities of transportation. The annual per capita consumption of meat has almost doubled during the past half century. It is estimated in pounds as follows:

United States	147
England	100
France	72
Germany	64
Russia	50
Italy	24[2]

Structure and Composition of Meats.—Meat is composed of muscular fibers, and the structures intimately associated with them, such as connective tissue, blood vessels, nerves, lymphatic vessels, and more or less adipose tissue.

The toughness of meat is due to the thickness of the walls of the muscle tubes and excess of connective tissue which binds them together, hence the flesh of young domesticated animals is usually more tender than that of old or wild animals.

The flavor of meat varies with the animal's age, its food, breed, and condition when killed. The meat of male animals, excepting pigs, is usually more highly flavored than that of females.

Meat contains albuminoids and gelatinoids; the latter through ac-

[1] "Statistical Account of the British Empire," Vol. II, p. 502.
[2] Thompson. "Practical Dietetics."

tion of hot water or steam are converted into gelatin. In addition meat contains the following nitrogenous substances: syntonin, myosin, muscle albumin, serum albumin, and numerous extractives, such as creatin, creatinin, xanthin, hypoxanthin, lactic acid; and small quantities of inosit and glycogen.

Meat at once after slaughter has an alkaline reaction, is tough, and possesses a sweetish and rather unpleasant flavor. Rigor mortis soon sets in, accompanied by the following changes: the reaction of the meat turns acid, owing to the development of sarcolactic acid; the connective tissue and fibers are softened as the result of autolytic enzymes and also as a result of bacterial action. While the meat becomes more tender, it also develops pleasant flavors. It is, therefore, not advisable to use meat at once after slaughter, but it should be allowed to hang at least two or three days. It is important during this time to preserve the meat from contamination with pathogenic microorganisms and to retard the growth of the saprophytes.

Nutritive Value of Meat.—The nutritive value of meat depends mainly upon the presence of proteins and fats. Nitrogenous extractive matters, such as creatin, xanthin, etc., sometimes called meat bases, are formed by cleavage of the proteins, but are of little value as foods. These nitrogenous extractives are present in about the same amount in both red and white meats, with the single exception of venison, which contains the least amount.

Meat must be regarded as a condensed and expensive food. For instance, a steak that costs twenty-five cents a pound contains over one-third or one-half of inedible substances, so that the edible portion really costs double that amount. On the contrary, when a pound of flour or cereal is purchased, the price of which is perhaps only one-eighth that of meat, the whole of it is edible.

Beef extracts are nothing more or less than a soup or soup stock specially prepared from beef. They first became generally known through the researches of Liebig, and are now an important article in commerce. The composition of the ordinary beef extract of commerce contains from 15 to 20 per cent. of moisture, from 17 to 23 per cent. ash, and from 50 to 60 per cent. of meat bases. These meat bases are the soluble nitrogenous contents of meat. They contain only a trace of soluble albumin, albumoses, and peptone. The chief meat bases which form the principal part of the substance are creatin, creatinin, xanthin, carnin, and carnic acid. It is, therefore, evident that meat extracts contain little nutritive matter, although this, being in a state of solution, is probably more readily absorbed than a similar amount of other nutritives in the form of ordinary meat. Wiley properly points out that the claim made by manufacturers is misleading, in that one pound of extract contains the nutritive properties of many pounds of

meat. Such a statement is absurd upon its face, and should not be allowed to go unchallenged. These extracts may be useful as stimulants or as condiments, or as a means of speedily introducing a soluble nutrient in the case of disease, where it is extremely important that even small amounts of nutritious material should enter the body.

A distinction should be made between beef extract and beef juice. *Beef juice* is obtained by strong pressure and is concentrated *in vacuo* to the proper consistence, or it may be used freshly prepared in the household.

Sources of Meat.—The principal source of meat is from cattle, sheep, and swine. In many places the flesh of horses, dogs, and cats is eaten. In Germany horses and dogs are slaughtered and regularly inspected for human food. The meat of these animals is also used in other countries that have long been flesh hungry. There is no sanitary objection to the use of such meat. Horse meat, when eaten in ignorance of its true character, makes no unpleasant impression. In Paris, Vienna, and other cities large quantities of horses, mules, and donkeys are slaughtered for food. Even in the United States several thousand horses are slaughtered and officially inspected each year with other food animals. It was formerly difficult to distinguish horse meat, but this is now rendered comparatively easy by means of the specific precipitins. (For this test see page 400.)

The different kinds of meat may be detected by physical, microscopical, chemical, or biological tests. Ordinarily meats from different animals may be distinguished by their odor or taste. Microscopically the fibers resemble each other so closely that this test is not to be relied upon. Meat varies somewhat in chemical composition from different species, from different animals of the same species, and even from different muscles in the same animal. The principal difference in the chemical composition of meats from animals of different species consists in the glycogen and fat content. Thus, horse meat contains considerably more glycogen than beef. The glycogen test, however, is not reliable because it may be changed as a result of bacterial action.

The fats of different animals have different physical and chemical characteristics. The fats crystallize in different forms and have different melting points; also the fatty acids derived therefrom. A careful examination of the fat, therefore, will lead to an approximate degree of knowledge concerning the character of the flesh from which it has been derived. For instance, lard and beef fat are easily distinguished from each other.

The Recognition of Spoiled Meat.—The recognition of spoiled meat that is also injurious to health is a very difficult task. Meat that is decomposed, putrid, or offensive, and thus objectionable to the senses, needs no further condemnation. The most serious infections and pois-

ons in meat, however, do not, as a rule, affect its appearance, odor, or taste, or do so so slightly as readily to pass unnoticed. Certain putrefactive changes brought about by bacterial action, which give the high or gamy taste so much prized by epicures, appear not to be injurious. Dogs and other carnivora prefer putrid flesh.

Meat inspectors are usually instructed to condemn meat that has not a red, fresh appearance, especially if it has become brownish or greenish. The meat is to be condemned if, upon pressure, much fluid of abnormal color or alkaline reaction exudes; if the fat is not yellow and firm, especially if soft and gelatinous; if the marrow of the femur is not firm and rose-colored and has become soft and brownish. Spoiled meat under the microscope shows obscurity of the cross striations of the muscle fibers and numerous bacteria. For a further discussion of this subject see Meat Inspection, and also the various diseases which render meat unsuitable or injurious as food.

Prevention.—The prevention of infections and poisoning from meat and meat products depends, first of all, upon the health of the animal, next upon the mode of death, and finally the methods of butchering, preserving, and handling the flesh. Careful attention to every detail is necessary all along the line. Cleanliness approaching surgical methods on the part of the butcher during the preservation, transportation, and preparation of the meat is called for. A careful system of meat inspection is a good sanitary safeguard. Thorough cooking is the most important protection we have against infection.

Meat should never be eaten raw, even where there is a carefully conducted inspection by trained experts. Individual cysticerci (tapeworm larvæ) are very easily overlooked, and one is enough to bring forth a tapeworm. It is also not possible to examine all hogs, particularly those slaughtered in country districts, for trichina, and even where this is done with care the method does not afford complete protection. It should further be remembered that some of the more serious bacterial infections do not alter the color, taste, or appearance of the meat in any way. Raw meat does not have a higher nutritive value than cooked meat, and is no more easily digested.

Special measures of prevention will be discussed under each infection.

Meat Preservatives.—The regulations of the U. S. Department of Agriculture permit the addition to meat or meat food products of the following substances: common salt, sugar, wood smoke, vinegar, pure spices, saltpeter, benzoate of soda. Only such coloring matters as may be designated by the Secretary of Agriculture may be used.

The adulterants most commonly used in meats are saltpeter, boracic acid, borax, sulphite of soda, and benzoic acid.

MEAT INSPECTION

The necessity for a careful sanitary control of our food is growing greater year by year in order to protect the consumer. This is especially necessary in the case of animal food products, especially meat and milk, which are most apt to carry infections and are the most readily decomposable. The necessity for this inspection is accentuated by the fact that the producer and the consumer are often separated by great distances, and, further, there are several middlemen between the two. The ignorance or greed of the middleman or the producer may force upon the consumer meat that is injurious or that is considerably below value.

The danger does not consist alone in eating infected or decayed animal products; the mere handling of flesh of some animals having had anthrax or glanders may be sufficient to transmit infection to the butcher or housewife, who may injure themselves in cutting the meat. An efficient meat inspection system is not only of advantage to man, but is the means of detecting and preventing disease among cattle, sheep, and swine. A sharp outlook at the slaughter house will discover the first appearance of rinderpest, foot-and-mouth disease, Texas fever, or other epizootic, which may then be quickly traced to its origin and nipped in the bud.

The border line between health and disease is ill-defined. It is doubtful whether any animal slaughtered for food is wholly sound and free from disease. Parasitic infections among the lower animals are exceedingly common. Anyone may convince himself of this fact by a visit to a slaughter house, for there he will see that many hogs have a handful of round worms in the intestinal tract; most animals have one or more species of intestinal worms, such as hookworms, tapeworms, and many protozoa, but, fortunately, these are for the most part not dangerous to man. Almost every hog or beef that is killed contains a *sarcosporidia*, a small parasite that inhabits only the muscles of these animals and which is harmless to man. It is, therefore, at once evident that the line in meat inspection cannot be drawn between health and disease, but aims to eliminate those diseases which are injurious to man and those diseases and conditions which render the meat of inferior quality or otherwise unfit for use. In establishing correct principles to guide a meat inspection service sentiment must give way to science. The killing of animals and the dressing of the carcasses is not a kid-glove business. In our country much good meat is condemned and destroyed according to law as a result of supersensitiveness. When meat becomes scarcer and prices higher this waste will be checked by a closer adherence to a sound

application of pathology. McCabe estimates that the value of the carcasses or parts of carcasses destroyed for food by federal inspection during the course of one year is more than $2,500,000. Dyson places the loss at about three million to three and a half million annually.

The principles of meat inspection vary in different countries, depending upon the local conditions. Thus, in Germany and other European countries, which have long had a scarcity of meat, and the people are, therefore, flesh hungry, much meat is passed for food that would here be condemned. In countries where meat is not very abundant it is even necessary for the officials to keep a sharp watch to prevent the people from eating meat known to be injurious. In America our attitude is very different, for we have a repugnance even against meat known to contain a harmless parasite. The records of the sanitary sciences are full of illustrations of the consumption of meat from animals known to be diseased.

A meat inspection service should have for its object first of all the protection of the consumer against diseased or other injurious qualities contained in the meat. This should be accomplished with as little waste of food products as practicable, and, finally, the meat should be so labeled that the consumer may know just what he is buying.

The Abattoir.—So long as animals are permitted to be slaughtered in any barn or cellar it is impossible to exercise a proper control over meat and meat products, and filthy conditions which endanger the public health will prevail. The first essential of a good meat inspection service is to concentrate all slaughtering in large central sanitary abattoirs. This simplifies the inspection and sanitary control, and is a needed measure to protect the consumer. In Germany and England public abattoirs have been established which belong to the city. These structures are built thoroughly of brick and concrete, and they are well protected against rats. They are situated near a railroad, so as to facilitate transportation, and are so constructed that they may be kept clean. Each person who wishes to slaughter must obtain a permit and pay rent. In the entire city of Paris there are only three slaughter houses. The erection and maintenance of sanitary slaughter houses is one of the needs of our country, especially in the smaller towns, and until this reform is accomplished we shall never have a satisfactory solution of the meat problem.

An abattoir must be especially well constructed and kept clean. The same may be said of the trucks, drays, and all objects that come in contact with the meat. The water-closets, toilet-rooms, and dressing-rooms should be entirely separated from the departments in which the carcasses are dressed or meat products handled or prepared. Attention must be paid to eliminate all sources of odor that may contaminate the meat, and every effort must be made to keep out flies

and other vermin, especially rats and mice. Dogs should not be allowed around slaughter houses on account of the danger of trichinosis and other parasites. The feeding of hogs on the refuse of slaughter houses should not be permitted.

The employees themselves must be cleanly and should wear clean outer clothes that may be readily laundered. The federal regulations even prescribe that employees shall pay particular attention to the cleanliness of their boots and shoes. It is just as important to wash the hands before beginning work, and to be particular after each visit to the toilet in the slaughter house or butcher shops, as it is in the milk industry. Persons with tuberculosis or other communicable disease should not be permitted in any department of the work where the meat or meat products are handled or prepared in any way. It is important that butchers who handle a diseased carcass should thoroughly cleanse their hands of all grease and then immerse them in a good disinfecting solution. Butchers' implements used on diseased carcasses should be sterilized in boiling water or strong carbolic acid or formalin solution and thoroughly cleansed before they are again used. The federal meat inspectors are required to furnish their own implements for their own dissection or examination of diseased carcasses or unsound parts. The precautions required in an abattoir and butcher shop are based on the same principles necessary in a surgical clinic. Meat that falls upon the floor or otherwise becomes soiled is required to be removed and condemned. Inflation by air from the mouth should not be permitted, though inflation by mechanical means is allowed by the federal meat inspection regulations. Only good, clean, and wholesome water and ice should be used in the preparation of the carcasses, and the wagons and cars and all surfaces with which the meat comes in contact should be kept clean and in good sanitary condition. There is no objection to the use of the skin and hoofs of animals condemned for tuberculosis and other diseases (except anthrax) communicable to man, provided they are disinfected. Each skin and hide must be immersed for not less than five minutes in a 5 per cent. solution of aqua cresolis compositus or a 5 per cent. solution of carbolic acid or a 1-1,000 solution of bichlorid of mercury.

Every complete abattoir must be provided with a retaining room, a condemned room, and a tank room. The retaining room is a separate compartment set apart for the final inspection of all carcasses and parts which the inspector desires to examine more carefully at his leisure. The retaining room must be large enough to have carcasses hang separately, furnished with abundant light, and provided with sanitary tables and other necessary apparatus. The condemned room must be securely ratproof and be under the lock and seal of the inspector. The object of this room is to contain all carcasses and

parts of carcasses until they can be tanked or disposed of in accordance with instructions.

All condemned carcasses or parts of carcasses are tanked under special requirements in an official abattoir. Tanking consists in exposing the carcasses to steam under a pressure of not less than 40 pounds, producing a temperature of 228° F., and maintained not less than six hours. This effectively renders the contents of the tank unfit for any edible product. In the absence of tanking facilities the condemned meat may be slashed with a knife and then denatured with crude carbolic acid, kerosene, or other agent, when it may be removed to some other establishment having proper tanking facilities.

Qualifications of a Meat Inspector.—A corps of thoroughly trained meat inspectors is one of the most important links in the chain of an efficient meat inspection system. A meat inspector should be a qualified veterinarian, having special experience and training for his specialty. He must know the anatomy of the various food-producing animals, especially cattle, horses, swine, sheep, and also fowl, and must be acquainted with the normal parts of each. He must be able to distinguish between the various organs of the various species, so that he cannot be imposed upon by those who would like to substitute one for another. He must know how to examine animals during life, in order to determine whether they are healthy. He must know the character of all the infectious diseases which are likely to pass through the district where he is situated. The government recognizes that it requires a high degree of skill to conduct this work, and it has, therefore, placed the meat inspection service under the Civil Service, and, further, will admit veterinarians only if graduates of recognized veterinary colleges. In addition they are required to pass a Civil Service examination.

The Freibank or Three-Class Meat System.—In Germany and certain other European countries meats are divided into three classes, viz., a first class, including meats which are passed for unrestricted trade; a second class, or Freibank meats, including meats which are allowed on the market under certain restrictions; and a third class, including meats which are condemned and thus excluded from the food supply.

The federal meat inspection system of our country is essentially a two-class meat system, that is, meats coming to inspection are either passed for unrestricted trade or they are condemned and thus excluded from use as food.

The system of the German *Freibank* and the compulsory declaration of the condition of inferior meats are very old. The municipal laws of Augsberg as long ago as 1276 prescribed that inferior meat should not be sold without giving notice as to its quality. In 1404 the mu-

nicipal laws of Wimpfen provided that the *Freibank* (from the German *frei*, free, here in the sense of unconnected or separate, and *bank*, a counter or stall) should be situated three paces away from the regular counters. The *Freibank* is, therefore, a counter which is free or separate from the counters on which the first class meats are sold. The term *"Finnenbank"* is sometimes used for these special meat stalls because measly meat or *"finneges Fleisch"* especially is sold at these places. This system of the *Freibank* has been extended quite generally in Germany and is rapidly extending in France, Belgium, Italy, and other European countries. Meat from tuberculous animals, from animals containing cysticerci (the larval stage of tapeworms), trichinous meat, and meat that would otherwise be injurious if eaten raw, but is entirely safe as far as these infections are concerned when thoroughly cooked, is first sterilized by steam before it is placed upon the *Freibank*. It has been the more or less general experience that the introduction of the *Freibank* system has at first been met with by prejudice from various sides, but it is also the experience that this prejudice gradually wears off, and that in some places the demand for this meat becomes greater than the supply. In any event, no large quantity of such meat should be sold to any one purchaser, so as to prevent its being used to any great extent in boarding houses and restaurants.

Emergency Slaughter.—In Germany the system known as emergency slaughter or *Nothschlachtung* has developed to large proportions. Animals that are sick or injured are killed, examined, and, if suitable for food, are labeled, inspected, and passed. In this way much valuable foodstuff is saved that would otherwise be lost. It is said that over 1 per cent. of the animals killed for food in Germany come under this emergency rule. There is also a certain amount of what may be termed emergency slaughter going on in the local uninspected slaughter houses of America, but it is not countenanced by the law, and is, therefore, done in secrecy. In this way the consumer buys inferior meat of third or fourth quality often at first class prices. In Germany the meat of animals killed under the emergency laws is so labeled and sold as second quality.

Methods of Slaughter.—In slaughtering, the principal indications are: (1) a sudden and painless death; (2) an immediate withdrawal of the blood; (3) removal of intestines and hair or hide; (4) immediate cooling. Animals should be kept without food for at least 12 hours before slaughter. Sheep and hogs are usually hung by the hind feet and the large vessels of the neck dexterously cut with a sharp knife and with a single motion of the hand. Cattle are usually first stunned by a blow upon the head, then hung up by the hind legs and bled.

The Jewish method of slaughtering is regarded by many as su-

perior to any other. It consists in cutting all the large vessels of the neck with one cut of a long, keen knife. The method is part of a ritual which includes an inspection of the animal and its organs for evidence of disease, according to the Mosaic laws. This is the oldest system of meat inspection. According to Dembo[1] it is the most rational from a hygienic standpoint, since the animal is bled rapidly and completely, and the convulsive movements cause the meat to be more tender and of more attractive appearance. Rigor mortis comes on more quickly, and the meat is, therefore, more quickly available for use, and also will keep several days longer than ordinarily.

A process of slaughtering originating in Denmark appears to have borne the test of trial in a very satisfactory manner, and recommends itself for adoption in the tropics, where meats decompose with exceeding rapidity. The animal is shot in the forehead and killed, or stunned, and as it falls an incision is made over the heart and the ventricle is opened for two purposes: to allow the blood to escape and to admit of the injection of a solution of salt through the blood vessels by the aid of a powerful syringe. The process requires but a few minutes, and the carcass may be cut up at once.

The common methods of killing fowl intended for the market are either by bleeding, by dislocation of the neck, or by chopping off the head. When the neck is stretched and dislocated the skin remains unbroken and no bruised effect is produced, but most of the blood in the body drains into the neck and remains there. In killing a fowl by bleeding the common procedure is to string it up by the legs with the head hanging downward. The operator then gives it a sharp blow with a stick on the back of the head, and when he has stunned it by this means he inserts a sharp knife into the roof of the mouth, penetrating the brain. He also severs the large vessels of the throat by rotating the knife, and the bird rapidly bleeds to death.

The United States Meat Inspection Law.—The Federal Meat Inspection Law, approved June 30, 1906, provides for the inspection of cattle, sheep, goats, and swine, the meats or meat food products, which are to enter into interstate or export trade. It is administered by the Bureau of Animal Industry under the direction of the Secretary of Agriculture. It should be remembered that the Federal Meat Inspection Law applies only to meat and meat products sold in interstate commerce or for export trade, and does not apply to meats butchered, dressed, and sold within the state. In accordance with our dual form of government, the inspection of meat that is slaughtered, dressed, and sold within the borders of a single state is left entirely to the authority of that state. It is not until some of this meat passes the state line that

[1] *Deutsche Vierteljahresschrift für öffentliche Gesundheitspflege,* XXVI, p. 688.

it enters interstate traffic and comes under the provisions of the federal law. Some of the states have passed laws similar to the federal law to protect their own citizens. In this way a more or less uniform method of meat inspection is gradually extending throughout the country.

The federal law provides for the inspection of the slaughter houses, the packing houses, the meat-canning, salting, rendering, or similar establishments; for the inspection of animals before and after they are slaughtered and for the condemnation and destruction of diseased carcasses or parts of carcasses. It also takes cognizance of the sanitary conditions of the establishments and the health of the employees. The carcasses are either passed and labeled "inspected and passed," or condemned in whole or in part to the tank, where they are steamed or immersed in strong sulphuric acid and reduced to inedible grease.

Ante-mortem Inspection.—A careful ante-mortem examination or at least an inspection of all cattle, sheep, swine, goats, etc., about to be slaughtered should be made by a competent veterinarian. Any animal showing symptoms of or suspected of being infected with a disease or condition which would probably cause its condemnation when slaughtered should be set aside. These animals should then be slaughtered separately in a place provided for this special purpose. If necessary the temperature of the animal may be taken in the ante-mortem examination, although due allowance must be made for rise in temperature due to excitement and undue exertion, especially in hogs. Animals commonly termed "downers" or crippled animals are set aside and slaughtered separately.

Post-mortem Inspection.—The post-mortem inspection is nothing more or less than a well-conducted autopsy. The head, tongue, tail, thymus gland, and all viscera, and also the blood and all parts used in the preparation of food and medicinal products should be retained in such a manner as to preserve their identity until the post-mortem examination is completed. It is, of course, impracticable to formulate rules to cover all conditions and diseases, and much must, therefore, be left to the judgment, experience, and training of the veterinary inspector in charge. Carcasses or parts of carcasses with the following diseases or conditions are condemned, depending upon circumstances: anthrax, pyemia and septicemia, vaccinia, rabies, tetanus, malignant epizootic catarrh, hog cholera and swine plague, actinomycosis, caseous lymphadenitis, tuberculosis, Texas fever, parasitic icterus, hematuria, mange or scab, trichinosis, tapeworms, infections that may cause meat poisoning, icterus, uremia, and sexual odor, urticaria, melanosis, tumors, bruises, abscesses, liver flukes, and other parasites, emaciation from anemia, milk fever, and railroad sickness. A few of these diseases deserve brief mention.

Tuberculosis.—Tuberculosis is exceedingly common in cattle and is becoming more and more prevalent among hogs. A preponderating percentage of all carcasses condemned as unfit for food is so condemned on account of tuberculosis. Thus, about 0.5 per cent. of all hogs slaughtered at Boston are condemned, but of these over 95 per cent. are condemned for tuberculosis. Tuberculosis is important, not alone because so many food animals are infected with it, but because it presents a peculiarly difficult problem for the meat inspector. The fundamental thought in determining whether to pass or condemn meat of a tuberculous animal is that it should not contain tubercle bacilli, and should not be impregnated with toxic substances of tuberculosis or associated with septic infection. If the lesions are localized and not numerous, if there is no evidence of distribution of tubercle bacilli throughout the blood, and if the animals are well nourished and in good condition, there is no reason to suspect that the flesh is unwholesome, and it is permitted to be used after the removal of the infected portions. Just when tuberculosis should be considered localized or generalized, from the standpoint of meat inspection, is frequently a difficult question to determine. Fortunately, the danger from this source is not very great, as tuberculosis of muscle is exceedingly rare, and the further safeguard of cooking is sufficient to kill the tubercle bacilli, provided the meat is thoroughly cooked throughout. The relation of bovine tuberculosis to human tuberculosis has been discussed on page 124.

Tuberculosis of cattle shows itself in four primary lesions: (1) the retropharyngeal lymph nodes, (2) the lungs and associated lymph nodes, (3) the mesenteric lymph nodes, and (4) the liver. From the retropharyngeal nodes the process extends to the cervical lymph nodes and also to the anterior mediastinal lymph nodes. When this group of glands alone is infected the disease may be considered as localized. From the mesenteric lymph nodes the infection frequently reaches the peritoneum, and from the bronchial lymph nodes the pleura. The newly formed growth in the peritoneal or pleural cavities may be enormous in amount. It is often suspended from the omentum in great grape-like masses (Perlsucht), or the intestines may be plastered with tubercles. In these cases the animal otherwise may be in good condition; that is, the disease is still outside the vital organs and the tubercle bacilli have not invaded the blood stream. In Germany it is permitted to cut off such growth and allow the meat to go into consumption. In our country the meat of such animals is rejected.

For practical purposes it is necessary to formulate definite rules for the guidance of the veterinary inspector, and this is done with minute particularity in the regulations of the Bureau of Animal Industry in the case of tuberculosis. In general, if the tuberculous lesions are limited to a single part or organ of the body without evidence

of recent invasion of tubercle bacilli into the general circulation, the diseased parts are removed and the remainder of the carcass is passed for use. If the animal suffered from fever before it was killed or is cachectic, anemic, and emaciated, or if the lesions are generalized, especially if they exist in two or more body cavities, or if the lesions are found in the muscles, intermuscular tissues, bones, or joints, or if the lesions are multiple, acute, and actively progressive, the carcass is condemned.

Anthrax.—All carcasses showing lesions of anthrax, regardless of the extent of the disease, are condemned and immediately incinerated. This includes the hide, hoofs, horns, viscera, fat, blood, and all portions of the animal. The killing bed upon which the animal was slaughtered must then be disinfected with a 10 per cent. solution of formalin, and all knives, saws, and other instruments that have come in contact with the infection must be boiled or otherwise disinfected.

Hog Cholera and Swine Plague.—Carcasses showing well-marked and progressive lesions of these diseases in more than two of the organs (skin, kidneys, bones, or lymphatic glands) are condemned. If the lesions are slight and limited they may be passed and the meat used for food.

Actinomycosis.—If the animal is in a well-nourished condition and the disease has not extended from a primary area of infection in the head, the head, including the tongue, is condemned and the remaining part of the carcass may be used, but if the disease is generalized the entire carcass is considered unfit for human use and condemned.

Tapeworm Cysts.—Carcasses of animals affected with tapeworm cysts, known as *Cysticercus bovis* and *Cysticercus cellulosæ,* are rendered into lard or tallow unless the infection is excessive, in which case the carcass is condemned. Carcasses or parts of carcasses found infected with hydatid cysts (echinococcus) may be passed after condemnation of the infected part or organ.

Septic and Pyemic Conditions.—All carcasses of animals so infected that consumption of the meat or meat food products thereof may give rise to meat poisoning should be condemned. For the information of the inspector the following conditions are specified: (1) acute inflammation of the lungs, pleura, peritoneum, pericardium, or meninges; (2) septicemia or pyemia, whether puerperal or traumatic or without any evident cause; (3) severe hemorrhagic or gangrenous enteritis or gastritis; (4) acute diffuse metritis or mammitis; (5) polyarthritis; (6) phlebitis of the umbilical veins; (7) traumatic pericarditis; (8) any other inflammation, abscess, or suppurating sore if associated with acute nephritis, fatty and degenerated liver, swollen soft spleen, marked pulmonary hyperemia, general swelling of the lymphatic glands, and diffuse redness of the skin, either singly or in combination.

DISEASES AND CONDITIONS FOR WHICH CONDEMNATIONS WERE MADE ON POST-MORTEM INSPECTION FISCAL YEAR 1910
(UNITED STATES)

Cause of Condemnation	Cattle		Calves		Swine		Sheep		Goats	
	Carcasses	Parts	Carcasses	Parts	Carcasses	Parts	Carcasses	Parts	Carcasses	Parts
Tuberculosis	27,638	48,997	184	166	28,882	720,775	.....	.....	.....	.....
Actinomycosis	527	53,008	1	85	.....	.....	.....	.....	.....	.....
Caseous lymphadenitis	.....	.....	.....	.....	.....	.....	1,122	.....	25	.....
Hog cholera	.....	.....	.....	.....	7,677	.....	.....	.....	.....	.....
Tumors and abscesses	171	7,070	35	61	932	1,516	164	41	3	.....
Septicemia, pyemia, and uremia	1,027	.....	309	.....	5,561	.....	539	.....	11	.....
Pregnancy and recent parturition	209	.....	.....	.....	40	.....	72	.....	.....	.....
Immaturity	.....	.....	3,472	.....	.....	.....	.....	.....	.....	.....
Pneumonia, pleurisy, enteritis, hepatitis, nephritis, metritis, etc	1,872	.....	346	.....	4,502	.....	1,572	.....	54	.....
Icterus	74	.....	43	.....	1,248	.....	909	.....	13	.....
Texas fever	435	.....	657	.....	.....	.....	.....	.....	.....	.....
Injuries, bruises, etc	3,333	5,253	499	166	383	2,915	657	183	11	1
Sexual odor	.....	.....	.....	.....	786	.....	.....	.....	.....	.....
Asphyxiation	.....	.....	.....	.....	630	.....	42	.....	.....	.....
Emaciation	6,476	.....	1,762	.....	932	.....	5,376	.....	81	.....
Miscellaneous	664	7,839	216	22	866	1,623	674	24,490	28	.....
Total	42,426	122,167	7,524	500	52,439	726,829	11,127	24,714	226	1

It is required that, immediately after the slaughter of any animal so diseased as to require its condemnation, the premises and implements used must be thoroughly disinfected. The part of any carcass coming in contact with the carcass of any diseased animal or with the place where such animal was slaughtered, or with the implements used in the slaughter, before thorough disinfection has been accomplished, should also be condemned. These infections are apt to give rise to meat-poisoning in those who eat the flesh of such animals. On account of the importance of this subject it will be discussed separately.

MEAT POISONING

Meat poisoning is almost always due to the presence and activity of certain bacteria belonging either to the paratyphoid or the hog cholera group. The meat may be infected as a result of disease in the animal before slaughter, or it may be contaminated post mortem from soiled hands, butcher's tools, rags, paper, dust, or other objects that come in contact with it. Chopping obviously favors the spread of bacteria throughout the mass. The inward growth of bacteria is greatly accelerated if the meat is kept warm. This may be prevented to a certain extent, or at least delayed, by drying the surface and by refrigeration.

Animals suffering during life from puerperal fever, uterine inflammations, navel infection (in calves), septicemia, septic pyemia, diarrhea, and local suppurations are apt to furnish meat containing the paratyphoid bacillus or closely related bacilli. Such meat has frequently given rise to meat poisoning. The bacilli causing meat poisoning may also invade the tissues of the animal as a terminal infection, and thus become dangerous to man.

In 1888, while investigating a large outbreak of meat poisoning, Gärtner isolated from the suspected meat and from the spleen of a patient who died a bacillus which he called *"B. enteritidis."* Ten years later Durham in England, and DeNobele in Belgium, independently isolated from cases of meat poisoning, and also from the meat, a bacillus closely allied to Gärtner's bacillus.

In 1896 Acharde and Bensaude isolated from the urine of a case of apparent enteric fever, and also from a purulent arthritis, following a similar illness, a bacillus which they called the "paratyphoid bacillus." In 1900-01 Schottmüller obtained from the blood of patients whose symptoms were those of enteric fever two bacilli resembling the paratyphoid bacillus of Acharde and Bensaude. These two organisms were named by Brion and Kaiser *"paratyphosus A"* and *"paratyphosus B."*

The relationship between these organisms was not at once recognized, but it has been shown during the last few years that they are

very closely allied to each other, and also to the hog cholera bacillus, *B. cholera suis*[1] of Salmon and Smith, discovered in 1885.

In fact, these bacilli belong to a group of organisms which has the typhoid bacillus at one end and the colon bacillus at the other. The intermediate forms in this group comprise the paratyphoid bacilli, the dysentery bacilli, the hog cholera bacillus, the *Bacillus psittacosis* (a disease of parrots communicable to man), the *Bacillus icteroides* (once associated with yellow fever), the *Bacillus typhi murium*[2] (the bacillus of mouse typhoid, the type of all the bacterial rat viruses), the *Bacillus enteritidis* of Gärtner (associated with meat poisoning and diarrheal diseases), the *Bacillus paracolon* of Buxton, the *Bacillus pseudotuberculosis rodentium* of Pfeiffer, and others. The organisms comprising this group are so closely related that it is often difficult to determine where specific differences begin and terminate. This group may be taken as a beautiful instance of missing links, and a study of these closely related organisms excites the imagination to the belief that we may here see evolution in the making.

Bainbridge[3] regards meat poisoning and paratyphoid fever as distinct diseases caused by different but closely allied bacilli. He considers meat poisoning to be caused by the *Bacillus enteritidis* or *B. cholera suis*, while paratyphoid fever is caused by *Bacillus paratyphosus A* or *B.* Bainbridge further regards paratyphoid fever as spread mainly by human bacillus carriers, and not as an infection contracted from meat, whereas meat poisoning in his opinion results from the consumption of food derived from infected animals or food that is contaminated after slaughter. Bainbridge believes that, as a rule, the clinical picture is quite dissimilar, paratyphoid infection resembling typhoid fever, while meat poisoning usually resembles an acute gastroenteritis. He admits, however, that a reversal of the clinical picture sometimes occurs. Much further work will have to be done before the subject will be entirely clear.

Fischer divided meat poisoning into three clinical forms: (1) typhoidal, (2) choleraic, (3) gastroenteric. The chief seat of attack in true meat poisoning is the gastrointestinal canal; the local irritation is frequently followed by a general bacterial infection. On the other hand, sausage poisoning (*Bacillus botulinus*) expends its chief attack upon the nervous system.

Cases of meat poisoning vary greatly in intensity and also in their

[1] This microorganism is also known as *B. suipestifer.*

[2] In Japan and elsewhere cases of poisoning with bacterial rat viruses in man have been reported. Most of these cases were caused by preparing rice in the same bowl used for mixing the rat virus.

[3] Bainbridge, F. A.: "The Milroy Lectures on Paratyphoid Fever and Meat Poisoning." *The Lancet,* March 16, 1912, Vol. I, No. XI, p. 705, and two succeeding numbers.

clinical picture. The period of incubation in the acute gastroenteric type is usually short, rarely over 48 hours; the period of incubation in the cases resembling typhoid fever is generally from 8 to 18 days.

The symptoms usually caused by the acute form of gastroenteric meat poisoning are severe headache with rigor, speedily followed by nausea, diarrhea, vomiting, and abdominal pain. In some severe cases restlessness, extreme thirst, and nervous symptoms, such as cramp, become prominent. Coma may precede death. Fever is usually present; the temperature may reach 102° to 103° F. As a rule, the temperature falls to normal in from two to five days, and the symptoms subside. The total duration of the illness rarely exceeds a week, but convalescence is often delayed by general muscular weakness or by attacks of circulatory impairment.

Bainbridge studied forty outbreaks of meat poisoning caused by *B. cholera suis* or organisms resembling it. In twenty-eight of these the bacillus was obtained from the tissues, twice from the blood, eight times from the spleen and other organs, and in the remainder from the stools. In twenty-one cases it was obtained from the meat which was regarded as the cause of the outbreak. The illness was confined to those who ate the meat, though not every one who partook of it was ill.

Outbreaks caused by *B. enteritidis* of Gärtner, *B. cholera suis*, and their congeners are frequent. In Germany at least 260 outbreaks have been recorded during the years 1898 to 1908. Although Germany is preeminently the home of meat poisoning, outbreaks occur from time to time in most European countries and in America. They appear to be more frequent in countries in which uncooked meat is eaten. These outbreaks are more apt to occur in summer, when the bacteria have better chances of multiplying within the meat.

Hübener found that of 36 outbreaks 16 occurred during June, July, and August and 30 between May and October. In another series of 27 epidemics Sacquepée found that 11 occurred in June, July, and August and 20 between June and November.

The meat of cows and calves is most often responsible for meat poisoning, though that of horses, pigs, and goats has also been responsible. Dunham says that no known case has come from mutton, and that the pig has been implicated in only one outbreak which has been studied bacteriologically. This is of particular interest to bacteriologists, on account of the similarity between the hog cholera bacillus and the *Bacillus enteritidis*.

These infections are not only conveyed in meat and meat products, but also in other foods. Many an instance of so-called ptomain poisoning from salads, cream-puffs, ice-cream, etc., is probably nothing but

acute infections caused by one of the bacteria in the colon-typhoid group.

Paratyphoid Fever.—Paratyphoid fever both clinically and etiologically is a first cousin of typhoid fever. The two diseases are frequently indistinguishable at the bedside. It needs the aid of the laboratory to differentiate one from the other. Epidemiologically paratyphoid fever shows marked differences from typhoid fever.

Paratyphoid is a world-wide infection; epidemic outbreaks occur, but, as a rule, are of limited extent. Paratyphoid never occurs as great epidemic calamities, such as have been frequently observed in water-borne or milk-borne typhoid. Paratyphoid coexists with typhoid in endemic foci. Thus, in Washington somewhat over 1 per cent. of all the cases reported as typhoid fever were shown, upon bacteriological examination, to have been paratyphoid. In India the proportion is greater, being as high as 15 per cent.

The paratyphoid bacillus closely resembles the typhoid bacillus in its cultural and morphological characters. The principal cultural difference between the two is that the paratyphoid bacillus ferments dextrose, whereas the typhoid does not. They also vary greatly in pathogenicity for the lower animals. Typhoid cultures, as a rule, are not very pathogenic for the lower animals, whereas guinea pigs and mice are extraordinarily susceptible to paratyphoid cultures. Most strains will kill guinea pigs when 1/50 to 1/100 of a loop is injected into the peritoneal cavity. Rabbits are also susceptible; birds are entirely refractory; cattle, dogs, cats, hogs, sheep show a high degree of resistance to paratyphoid cultures.

A fundamental point of difference between the paratyphoid and the typhoid organisms is that they each have specific agglutinating properties. In any critical case this difference is the most important distinguishing feature. Care must be taken, in using agglutinins in differentiating these closely allied species, to guard against confusion through group agglutinins, and also to keep the proagglutinoid zone in mind.

The paratyphoid bacillus is a small rod with rounded ends and peritrichal flagellæ resembling the typhoid bacillus, except that it is more actively motile. It stains readily with anilin dyes, decolorizes by Gram's, does not liquefy gelatin, has no spore, and is a facultative aerobe; it clouds bouillon uniformly, and does not produce indol. Upon Endo's medium the paratyphoid colonies are pale, moist, translucent, with a bluish cast, quite similar to typhoid colonies.

The paratyphoid bacillus is divided into several subclasses. These classes vary with the classifier. Schotmüller in 1902 divided them into paratyphoid A and paratyphoid B. In the same year Buxton proposed that paratyphoid organisms be confined to those producing a disease

resembling typhoid, while paracolon be used to designate those organisms producing gastrointestinal disturbances. We now hear little of the paracolon group. Seiffert, who studied the question in Ehrlich's laboratory, considers two groups, viz., paratyphoid A, those producing permanent acidity in litmus whey, and paratyphoid B, those producing acidity terminating in alkalinity in litmus whey. Group B also produces characteristic lesions when injected into the muscles of pigeons. The group known as paratyphoid B is much more common and widespread than type A. It is interesting to observe that the agglutinins have a closer relationship between paratyphoid B and typhoid than between paratyphoid A and typhoid, although type B seems farther removed.

The paratyphoid bacillus may be found in the blood and internal organs, also in the feces; seldom in the urine. It produces a continued fever in man closely resembling typhoid fever. As a rule, paratyphoid is milder than typhoid. Lentz[1] gives a mortality of 3.3 per cent., against typhoid, which is about 9 per cent. Paratyphoid infections frequently manifest themselves as an acute gastroenteritis, having a sudden onset with vomiting, chill, and diarrhea and a sharp rise of temperature. Between these acute cases and the typhoid type there are all grades of severity and several clinical varieties.

Paratyphoid fever may be complicated with hemorrhages from the bowels, bronchitis, and pneumonic processes, just as in typhoid fever; relapses are rare. It is not definitely known how much of an immunity is conferred by one attack, but it is known that paratyphoid does not protect against typhoid, nor does typhoid protect against paratyphoid.

The paratyphoid bacillus, although it contains no spore, is claimed to have a higher degree of resistance to heat than the typhoid bacillus. Thus, Fischer[2] found that an exposure to 70° C. for 10 to 20 minutes did not destroy this microorganism. This unusual resistance is important, in view of the fact that the bacillus is usually conveyed in meat. As a rule, the inside of a large piece of meat or a bulky sausage does not reach 70° C. during the process of cooking.

The virus enters the body through the mouth and is discharged in the feces. The intestinal tract of man must be regarded as the great reservoir for paratyphoid infection, just as it is the main source of typhoid. The paratyphoid bacillus does not, as a rule, multiply in nature, except in meat, and perhaps other foodstuffs.

The paratyphoid bacillus does not necessarily exist in the tissues of the animal at the time of its death, but the meat may become in-

[1] *Centralblatt f. Bakt.*, Referate, Bd. XXXVIII.
[2] "Festschr. f. R. Koch," Jena, 1903.

fected while it is butchered or during any stage in its after-care. The paratyphoid bacillus deposited upon a roast, steak, or a carcass will grow readily and rapidly throughout the mass, especially if kept warm. It is easy to conceive how meat may thus become infected through the contamination of dirty hands, butchers' implements, soiled meat blocks, unclean cloths, etc. It is not unknown that carcasses or cut portions may fall upon the floor or in other ways be carelessly handled. This and other infections may also be carried to meat through flies, dust, and other well-known means of bacterial convection. Meat may be infected in the slaughter house, in the butcher shop, during transportation, or in the household. In fact, meat may become infected in very much the same ways that milk becomes infected, and the same care and cleanliness are called for in each case.

Paratyphoid fever is by no means always contracted from meat, but may be transmitted directly from man to man.[1] When this takes place the mode of transmission must, doubtless, be similar to that of typhoid fever.

The paratyphoid bacillus and the *Bacillus enteritidis* of Gärtner and all the other organisms belonging to this group, from the colon to the typhoid bacillus, grow well in milk. It is strange, however, that milk has never been incriminated as causing paratyphoid infection in man. The possibility is evident, but the occurrences have, perhaps, been overlooked.

Prevention.—Meat inspection affords but little safeguard against the meat poisoning group of bacteria, for the reason that these microorganisms may pervade the meat without in the least changing its appearance, color, flavor, or odor. Their presence may only be detected by bacteriological examination. In this respect paratyphoid and its congeners in meat correspond strikingly to typhoid and other infections in milk. Animals, however, suffering with septicemic infections, puerperal fever, metritis, diarrhea, and serious inflammations of any kind should be condemned by the meat inspector on account of the danger of conveying infections of the kind in question. Scrupulous cleanliness must be observed in slaughter houses, butcher shops, and the home. The butcher's hands and implements require cleanliness of a surgical order; housewives should refuse to patronize butcher shops that are not carefully screened, that do not have sufficient refrigeration, and that are not tidy and cleanly throughout. Greater care should also be exercised with meat in transportation, whether upon the railroad or the delivery wagon.

Meat that is hashed, as in Hamburg steak, or prepared in a sausage, is especially liable to infection on account of the additional handling and because the bacteria pervade the minced articles more readily

[1] Gärtner, v. Ermengen, Fischer, Bainbridge.

than they do the solid chunks. The paratyphoid bacillus has been isolated from sausages.

Thorough cooking destroys the infection and eliminates the danger of meat poisoning due to bacteria belonging to the group under consideration. It must not be forgotten, however, that meat after it is cooked and allowed to stand may become infected, and, further, that the cooking must be thorough.

Otherwise the prevention of paratyphoid fever corresponds in all essential particulars with that discussed under typhoid fever.

Botulismus or Sausage Poisoning.—Botulismus is a specific intoxication produced by a saprophyte. The symptoms are caused by a poison that is generated by the *Bacillus botulinus* outside of the body. The bacillus itself is harmless and does not grow and multiply within the body. In this respect botulismus is a sharp contrast to meat poisoning produced by the paratyphoid and related bacilli.

The *Bacillus botulinus* may grow and produce its toxine in sausage, meat, or fish, and occasionally in vegetables; in fact, it may develop upon protein media of all kinds. It requires time for the growth and development of the toxine; fresh foodstuffs, therefore, are not dangerous so far as botulismus is concerned. Meat, sausage, or fish that contains the botulismus toxine may or may not be altered in color, taste, or odor; sometimes the indications of putrefactive and fermentative changes are evident. But the presence or absence of such alterations are not in themselves an index of the presence or absence of the botulismus toxine.

Botulismus is more common in Europe than in this country. The first outbreak, studied by v. Ermengen, occurred in Ellezelles, Belgium, in 1895. Fifty persons were affected, of whom three died. The particular ham which was responsible in this case was preserved in a weak brine at the bottom of a cask under anaerobic conditions. Another ham in the same cask above the brine had a different bacterial flora and was not poisonous.

Sausages are the most frequent source of botulismus. The sausages readily become infected and present ideal anaerobic conditions for the growth of the organism, especially as they are rarely refrigerated and frequently contain old and contaminated scraps. The disease is, therefore, frequently called sausage poisoning from *botulus,* a sausage. Certain sausages, as, for example, the blood sausage and the liver sausage prepared in Württemberg and Baden, are especially apt to be infected. Salt fish, smoked ham, preserved meat, venison, old roasts have produced the intoxication. The bacillus may even grow in nitrogenous vegetables; thus, spoiled beans were responsible for an outbreak in Hessen.

Symptoms.—After a period of incubation of about 20 to 24 hours

there are nausea, gastric pains, vomiting, visual disturbances, such as fogging of the eyes, sometimes amounting almost to blindness, dilatation of the pupils, and loss of reaction to light, ptosis of both lids, and a peculiar stony stare. There are burning thirst and difficulty in swallowing, the mucous membrane of the respiratory tract is strongly reddened and covered with a thick viscid secretion, which causes severe attacks of coughing and even suffocation. There is extreme muscular weakness; the respiration and circulation remain normal; there is no fever; death may ensue; recovery is slow.

The *Bacillus botulinus* discovered by v. Ermengen in 1899 is a large, slightly motile rod, 4 to 6 μ long, 9 to 12 μ thick. It has slightly rounded ends; 4 to 8 flagellæ, generally single; rarely occurs in filaments; has a large polar spore; stains readily, and is Gram-positive.

The bacillus grows best between 20° and 30° C., but does not thrive above 35° C., hence does not multiply in the body. It is a strict anaerobe, and requires a distinctly alkaline medium. Its growth is favored by the addition of 2 per cent. grape sugar; gelatin is not liquefied, but active gas formation takes place similar to cultures of malignant edema and other anaerobes. It does not alter milk. All the cultures emit the odor of butyric acid. The spores are killed at 85° C. for 15 minutes or 80° C. in one hour.

The *Bacillus botulinus* has been isolated from various foodstuffs. Kempner obtained it from the feces and Landtman isolated it from poisonous canned beans. In habitat and biology it resembles tetanus spores, and it is, therefore, supposed to exist in the soil, but attempts to find it in the soil have resulted negatively. It is perhaps not widely distributed in nature.

Botulismus Toxine.—The bacillus produces a soluble, true toxine comparable in all respects to the poisons produced in cultures of diphtheria or tetanus. The poison, however, is produced outside of the body, not in it. There is no toxine production and little growth at body temperature. The bacillus is a true saprophyte; thus, the *Bacillus botulinus* occupies a position of its own, being pathogenic only by virtue of the poison formed on dead nitrogenous substances. It, therefore, is the type of a toxicogenic organism, a term proposed by v. Ermengen. The *Bacillus botulinus,* however, is the only definitely known example of this category.

There are doubtless other microorganisms that produce substances in dead organic matter which are poisonous when partaken of by man. Of these the *Bacillus proteus vulgaris* and the *Bacillus coli communis* (*Bacterium coli*) are believed to play such a rôle. Instances of poisoning with these organisms have been studied by numerous investigators, but their toxines and their action are less well understood than in the case of botulismus.

The botulismus toxine is the only one of the true toxines that is poisonous when taken by the mouth. It is thus pathogenic for guinea pigs, mice, and monkeys, as well as for man. One or two drops of a culture placed upon a piece of bread causes death in a few days. Toxines of diphtheria and tetanus are not poisonous when taken by the mouth. Cats and rabbits, however, withstand large doses of botulismus toxine by the mouth, but are very susceptible to subcutaneous or intravenous injection. The botulismus toxine is readily killed by heat, and is very susceptible to acids and alkalies; it also deteriorates in sunlight.

A true antitoxin may be obtained by injecting increasing amounts into susceptible animals. Kempner first obtained the antitoxin in goats. Much work has since been done upon this toxine and antitoxin by Madsen and others. The botulismus antitoxin has both protective and curative virtues in man as well as in experimental animals, even when given 24 hours after the ingestion of the poison. It has recently been prepared for distribution at the Institute for Infectious Diseases at Berlin.

Prevention.—Prevention of botulism consists in greater care and cleanliness in the handling and preservation of nitrogenous foodstuffs. There is no danger of this poison in fresh foods, or in foods preserved a long time, if properly sterilized in the can or properly refrigerated. The chief danger is from sausages eaten without cooking. The heat destroys the toxine, but it must be sufficient and penetrate throughout the mass; also the cooking must be recent, for the bacillus grows well in cooked foods. When obtainable the antitoxin may be used as a preventive in cases where a number of persons are showing symptoms of poisoning from having partaken of the same carcass or sausage or other suspected food.

Trichinosis.—*Trichinella spiralis*, formerly *Trichina spiralis*, is a round worm which passes its entire life cycle in man, rat, or hog. Many other animals, such as mice, foxes, guinea pigs, rabbits, cats, dogs, etc., are susceptible. This parasite differs from many other animal parasites in affecting several genera and in passing its entire life cycle in each host.

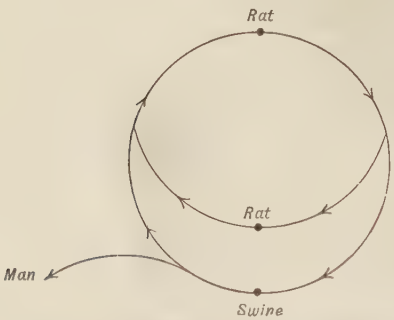


Fig. 73.—Trichinella Spiralis. Entire Life Cycle in Each Host.

The larvæ are imbedded in the muscles. When the trichinous meat is taken the capsules are dissolved in the stomach, the larvæ set free, the parasites enter the intestine, where they find conditions favorable, and where in a few days they become large enough to be seen with

the naked eye as fine threads, which grow into the full mature worm. The female parasite produces upward of five hundred young, and it is these embryos that pierce the bowel and penetrate into the tissues. They get into the blood stream and are thus distributed to the muscles. It takes about 7 to 9 days to accomplish these changes; that is, for the larvæ in the muscles to develop from the brood of embryos in the intestine.

The normal or common host of *Trichinella spiralis* may be regarded as the rat, which gets infected about slaughter houses and butcher shops. Hogs get the disease by eating rats, through feces, or directly from infected offal. Man receives the infection by eating trichinous pork (occasionally dog, cat, or bear meat).

Not all persons who eat trichinous flesh have the disease. A limited number of the embryos do not cause noticeable symptoms. In man the disease is well characterized in two stages: (1) gastrointestinal, (2) general infection. The symptoms of the second stage are fever, intense pain in the muscles caused by the migration of the parasites, edema, and leukocytosis. The count of the white cells may reach 30,000 with distinct eosinophilia.

The recognition of trichinosis as a distinct infection is recent (1860). The parasite was named by Richard Owen and was long regarded as harmless and as a curiosity. The infection was mistaken for typhoid fever, rheumatism, acute miliary tuberculosis, and other diseases of common occurrence. The particular case which finally revealed the parasite as being capable of harm was that of a young woman admitted to the hospital at Dresden suffering from a disease diagnosed as typhoid fever. The patient had agonizing pains in the muscles, and the autopsy revealed the parasite imbedded in vast numbers in the muscular fibers. An investigation led to the examination of some pork of which she had eaten four days before the first symptoms appeared, and showed the presence of the same parasite. Since then many local outbreaks have been described, more particularly in Germany, where the custom prevails of eating raw or underdone pork, especially in sausage.

Prevention.—The disease is practically never recognized in swine during life. The protection rendered by the inspection of meat is quite unsatisfactory. This inspection consists in compressing small fragments of the muscle (diaphragm, tongue, etc.) between two glass plates, which are then examined with a low power of the microscope for the encysted larvæ. That this examination is not an entirely satisfactory safeguard, even in cases where it is done with care and precision, is shown by the fact that in Germany, for example, the disease is still very common. The microscopic inspection of every carcass for trichina is expensive and open to several practical sources of error. It, there-

fore, gives a false sense of security and is impractical in country slaughter houses.

Our federal meat inspection regulations no longer require a microscopic examination of pork for trichina. Until recently all the pork dressed for export was examined by the microscopic method, but this has been discontinued.

To show the great prevalence of the disease, the microscopists of the United States Department of Agriculture found the parasite in 41,659 of the 2,227,740 hogs examined. The disease in man is probably more prevalent than the figures of the clinicians indicate. Careful search at autopsy showed that many persons have been infected but have recovered. Thus, Williams of Cleveland examined 505 cadavers and showed old encapsulations and calcifications in 27, or 5.34 per cent.

The trichinæ are not particularly resistant, being killed at 155° F. if they are not encapsulated, otherwise at 158° to 160° F.; that is, they have about the same resistance as non-sporulating bacteria. They are not affected by intense cold, putrefactive processes, nor by ordinary processes of cooking, but die after thorough cooking or a prolonged period of pickling. As the elimination of the disease in swine and rats will be a long and difficult campaign, the only satisfactory and absolute protection is complete cooking.

The rat should be regarded as the common reservoir of trichina, and a persistent warfare should be made against rats in slaughter houses, butcher shops, markets, and places where hogs are kept (see page 242. Human feces and contaminated offal must not be fed to hogs.

The Pork or Measly Tapeworm (*Tænia Solium*).—*Tænia solium* passes the larval stage of its life history in the flesh of pork. These encysted larvæ are known as bladder worms or *Cysticercus cellulosæ:* they are commonly called pork measles. Man eats these encysted larvæ which develop into adult tapeworms in the intestinal tract.

Infection with this tapeworm may be particularly dangerous, because the cysticerci have the peculiarity that they may occur in man as well as in the hog. When the cysticerci develop in important parts, such as the eye, brain, etc., death or serious consequences may ensue. The infection with this particular tapeworm is fortunately rare in the United States and Canada, but is more frequently met with in the old world. The adult tapeworm occurs only in man; the larva is found especially in hogs and occasionally in sheep and dogs. This parasite is smaller than the beef tapeworm. The head is armed with a double row of hooks, with which it maintains its hold to the mucous membrane. Each link contains a uterus with lateral branches, and the genital pore is marginal and irregularly alternate.

The source of infection in man is practically always undercooked or raw pork. Occasionally the infection is contracted from another person through the eggs in the feces. Hogs become infected from eating human feces containing the eggs, or from food and drink contaminated with them. To build a privy over the pig pen, as one sometimes sees in the country, means the formation of an endless chain in the biology of this worm.

Tænia solium produces less anemia than the fish tapeworm (*Dibothriocephalus latus*), but may be dangerous because of cysticercus formation in man. This is the only tapeworm in which this occurs. A person infected with *Tænia solium* may reinfect himself through dirty finger nails, unwashed hands, or other uncleanly habits, and it is also comparatively easy to infect others through the feces.

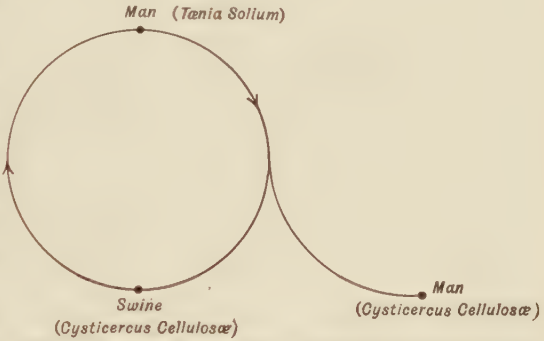


Fig. 74.—Tænia Solium, the Pork or Measly Tapeworm. Note that man may infect himself.

In prevention one must first consider the disposal of feces. Hogs heavily infected should be destroyed; those having a light infection may be thoroughly cooked and the meat eaten. Cold storage is not quite so effective in destroying the larvæ of *Tænia solium* as it is for *Tænia saginata*, for the former have been found alive after 29 days, whereas the latter die after 21 days.

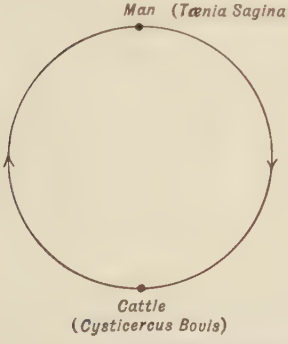


Fig. 75.—Beef Tapeworm.

Tænia Saginata.—*Tænia saginata,* also called *Mediocanellata,* occurs only in cattle and man. This tapeworm is rather common in our country, but is not dangerous, like *Tænia solium,* though at times it produces a certain degree of anemia and other symptoms. It is often difficult to expel, despite the fact that it has no hooks. In geographical distribution it is cosmopolitan. The adult worm occurs in man; the larva is found imbedded in beef and is known as the *Cysticercus bovis.* The uterus has 15 to 35 slender dichotomous branches on each side. The genital pore is marginal and irregularly alternate.

Man becomes infected by eating raw or underdone beef. The tongue and the muscles of mastication most frequently contain the larvæ.

Cattle become infected through the embryos passed in human feces, which contaminate their food or water.

Prevention.—The prevention depends first upon proper disposal of human excrement and an efficient system of meat inspection. The cysticerci die in three weeks after killing, hence meat that has been preserved 21 days may be regarded as safe. Proper cooking and thorough salting also kill the larvæ of this tapeworm.

FISH

Fish poisoning or ichthyotoxismus is most frequent in Russia, Japan, and the West Indies, and other seacoast countries in which fish forms a large part of the diet. Some of these poisons are now better understood, but for the most part are far from being satisfactorily explained.

Physiological Fish Poisoning.—Many fish are always poisonous, that is, normally contain a substance toxic to man; some develop the poison only during spawning season. Various species of the *tetrodon* and *diodon* frequently cause serious and fatal poisoning in Japan. In Tokio alone 680 fatal cases out of 933 were reported occurring in 1885-1892 from the so-called *"fugu."* In China and Japan such fish are sometimes taken for suicidal purposes. The active principle in fugu poisoning resembles curara. The poison is found in the ovaries and testicles, and is called "fugin." Its exact chemical nature has not been determined. The symptoms produced are: dyspnea, cyanosis, dilatation of the pupils, relaxation of the sphincters, paralysis of speech, dizziness, salivation, and vomiting. Death may result in one to two hours.

Few fish containing physiological poisons are found north of the tropics. Some fish, such as shad and smelts, are preferred during spawning season. However, during spawning season the roe of different members of the sturgeon family, of the pike, and the barbel have been known to cause pronounced and even fatal intoxication; the symptoms resemble those of gastroenteritis. It is interesting to note that even the codfish (*Gadus morrhua*), if eaten raw, has caused serious intoxication.

Bacterial Poisons.—Bacterial poisoning from fish is fairly common. The fish may be diseased, or when caught may be healthy, but the bacteria gain access and grow throughout the meat as the result of contamination or imperfect preservation. Bacterial diseases among fish are rather common and often occur as epizootics. In almost all the reported instances of injurious action resulting from bacteria the fish has been eaten raw. Bacteria may form poisonous substances in fish closely resembling botulismus. Fish caught by the gills in nets die slowly and decompose rapidly. They are of inferior flavor and

value and are more liable to be injurious than fish taken from the water and killed at once; under such circumstances they remain firm and retain their flavor longer than those that die slowly. In some parts of the world live fish in tanks are offered for sale in the markets. This procedure cannot be commended from a sanitary standpoint, for the tanks are apt to become dirty and the fish liable to sicken and die slowly, so that the object of purveying only live, fresh, and wholesome fish is largely defeated. It is well known that fish decompose readily and should, therefore, be handled in a cleanly manner and used as fresh as possible. When refrigerated the temperature should be low.

The Fish Tapeworm.—The principal animal parasite conveyed through fish is the tapeworm, *Dibothriocephalus latus,* which infects man wherever fresh fish forms a large part of the diet. The *cysticercus* or larval stage is found in the muscles and organs of various fresh water fish, particularly pike, perch, and several members of the salmon family, and when partaken of by man develops into the adult tapeworm in the intestines. The adult worm is also found in cats and dogs that feed upon fish.

The fish tapeworm produces a severe anemia resembling pernicious anemia. The head of the *Dibothriocephalus latus* is armed with hooks and attaches itself to the mucous membrane of the bowels. Faust and Talquist have shown that the anemia is due to an hemolytic action caused by oleic acid found in the head of the fish tapeworm. Each link of the fish tapeworm has a rosette-shaped uterus in the median line and a special uterine pore from which eggs are constantly discharged and may readily be found in the feces. It is through the pollution of the streams with sewage containing the eggs that the fish become infected.

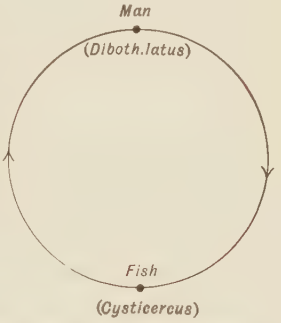


FIG. 76.—DIBOTHRIOCEPHALUS LATUS, THE FISH TAPEWORM.

Produces serious anemia.

The prevention of the fish tapeworm consists in proper disposal of feces, so as to prevent the contamination of fresh water streams, and the proper cooking of fish.

SHELLFISH

Shellfish include mollusks, as oysters, clams, mussels, and crustaceans, as lobsters, crabs, and shrimp. The conditions which render such food injurious are much the same as those discussed in connection with fish. Shellfish may be diseased when taken from the water, but little is known of the diseases of shellfish that influence man. Shellfish may be perfectly good and wholesome when fresh, but may become contaminated and poisonous on keeping, especially if not kept cold.

With shellfish, as with other foods, the intermediate products of putrefaction are the most dangerous. For example, mussels that have been allowed to decompose for some days have been shown to be free from toxic substances (see Ptomains). Perhaps the most important condition bearing upon the injurious properties of shellfish is their habitat. It has been shown repeatedly that, when grown or kept in polluted water, they acquire poisonous or infectious properties. On being transferred to fresh, clean water they may lose these injurious characteristics. It is claimed by some observers that 6 days, by others that 16 days, in clean water are sufficient for mollusks to purge themselves of typhoid infection.

It is now well known that oysters, and doubtless other mollusks, while in polluted water, may take up large numbers of different kinds of bacteria, and that these remain alive and virulent for a long time. Herdman and Boyce found 17,000 colonies from an oyster obtained from the neighborhood of a drain pipe. Ordinarily oysters from open waters contain less than 100 colonies. Oysters contain fewer bacteria during the winter months (December to March), when they probably hibernate. Oysters placed in polluted waters may retain the typhoid bacillus as long as 14 days after infection. Klein found typhoid to persist in oysters from 2 to 3 weeks. At times the oysters clean themselves in a week; this is facilitated by clear, clean, running water. The process by which the oyster rids itself of bacteria is perhaps both mechanical and biological.

Both typhoid and cholera have been convincingly traced to infected oysters. The oysters may become infected where they grow or during the process of "fattening" or "floating," which consists in soaking them in fresh water for the purpose of making them more plump and increasing their size. In the language of the fishermen, this is called "floating," "drinking," or "laying out." On account of the difference in osmotic pressure the water enters the cells of the oysters and certain mineral salts pass out. While the oyster increases in size and weight it is at the expense of the natural salt, mostly sodium chlorid, which the oyster contains.

It may be stated as a general rule that oysters and other shellfish should not be used when taken from water which, upon bacteriological examination, would render it unfit if used for drinking purposes. It should be remembered that bacteria found in mollusks represent the flora of the mud of the bed of the stream rather than the water itself. For instances of typhoid outbreaks attributable to oysters see page 91.

The prevention of typhoid and similar infections through oysters and other shellfish consists in regulating the location of the beds and in transferring doubtful ones to a clean situation in clear sea water until the bacteria have perished or have been washed away. How long

this may take is somewhat doubtful; perhaps a week, or, better, 16 days, should be allowed. "Floating" should be prohibited, especially in water of doubtful character. Thorough cooking will kill all the non-spore-bearing bacteria.

Mussel Poisoning.—*Mytilus edulis*, the common mussel, is a rather frequent source of poisoning in England and on the Continent. Three types are recognized clinically: (1) gastroenteric, (2) nervous, and (3) paralytic. In the first type the symptoms are nausea, vomiting, diarrhea, and sometimes choleraic symptoms. This form is similar to the common type of meat, cheese, and other acute food poisoning, and when due to mussel poisoning is not, as a rule, fatal. In the nervous type the symptoms are itching, erythema, urticaria, angina, dyspnea. Recovery from this form usually takes place in a few days. The paralytic type suggests curara. This is less frequent and more dangerous than the gastroenteric or nervous types. It may be compared to botulismus, but differs in rapidity of onset, the nature of the symptoms, and in the fact that boiling does not destroy the poison. Death has occurred in 15 minutes after eating boiled mussels. A notable example of mussel poisoning occurred at Wilhelmshaven in 1885. A large number of dock laborers and their families were poisoned shortly after eating cooked mussels; three died. The mussels were examined by Brieger, who isolated several basic substances or ptomains, one of which, mytilotoxin, was poisonous to animals, causing similar symptoms. Novy considers this a true instance of a heat-resisting alkaloidal-like poison or ptomain in a sense analogous to mushroom poisoning. Cats and dogs eating poisonous mussels suffer with symptoms similar to those seen in man, namely, paralysis, coma, and death. Rabbits have been poisoned by giving them the water in which the mussels have been cooked. Under these circumstances they may die in a minute.

Miscellaneous.—In addition to the infections noted, the following diseases are sometimes transferred from the flesh or organs of lower animals, or by contact with the lower animals in various ways: tuberculosis, anthrax, glanders, rabies, actinomycosis, foot-and-mouth disease, cowpox, ringworm, and various pyogenic and septic infections.

Meat may occasionally be injurious to health from a variety of miscellaneous causes. Thus, an animal that has died of arsenic or other poisonous substance may contain sufficient of the poison in the tissues to affect the person who eats part of the flesh.

"BOB-VEAL"

"Bob-veal" is the flesh of immature calves, that is, animals less than two or three weeks old. "Bob-veal" is objectionable from humanitarian

and esthetic grounds. The meat is flabby, edematous, soft. The connective tissue is gelatinous and is present in greater quantity than in mature animals. The fat is reddish-gray and soapy, the meat less nutritious in value, as it contains a large proportion of water. On account of its moist and soft condition "bob-veal" has a greater tendency to spoil than the flesh of mature animals. Young calves are highly susceptible to a number of infections, particularly diarrheal diseases, which enter through the infected navel. The flesh of such animals may convey microorganisms belonging to the paratyphoid group.

Ostertag states: "Putrefactive and pathogenic microbes find a ready media for luxuriant growth in 'bob-veal' carcasses. In Switzerland 27 persons became ill from eating veal of a calf five days old which had yellow water in the joints; one patient died. In the Grand Duchy of Baden from 1888 to 1891 5.3 calves out of every 1,000 slaughtered furnished meat injurious to health."

Bollinger recites: "In the epidemic at Andelfingen 450 people became ill and 10 died. The veal which was consumed was suspected and it transmitted its poisonous properties to beef stored with it. At Berminstorf 8 people died from eating veal from a calf four days old. At Morselle, Belgium, 80 people became sick from eating veal of two calves with diarrhea."

It is a well-known fact that calves under three weeks old have umbilical wounds which are liable to become infected. All young animals are subject to such infections, since nature is left to effect the healing of the wound. The weight of the calf is often taken as an indication of its age. Thus a calf weighing 40 pounds or more is considered mature, but the weight is a poor index of age. The condition of the umbilical wound usually tells the tale.

EGGS

Perhaps no article of diet of animal origin is more commonly eaten in all countries and served in a greater variety of ways than eggs. Eggs are used in nearly every household in some form or another. It has been calculated that on an average they furnish 3 per cent. of the total food, 5.9 per cent. of the total protein, and 4.3 per cent. of the total fat used per man per day. When we speak of eggs we ordinarily mean hen's eggs, but the eggs of ducks, geese, and guinea fowls are used to a greater or less extent; more rarely turkey's eggs and sometimes those of wild birds. Plover eggs are prized in England and Germany, while in this country the eggs of sea birds, such as gulls, terns, herons, and murres, have long been gathered for food. Other eggs besides those of birds are sometimes eaten. Turtle's eggs are highly prized in most countries where they are abundant. The eggs of

the terrapin are usually served with the flesh in some of the ways of preparing it for the table. Fish eggs, especially those of the sturgeon, are preserved in salt under the name of caviare. Shad roe is also a familiar example of the use of fish eggs as food. The eggs of alligators, lizards, serpents, and some insects are eaten by races who lack the prejudices of western nations.

Hen's eggs vary considerably in size and appearance. The shell constitutes about 11 per cent., the yolk 32 per cent., and the white 57 per cent. of the total weight of the egg. The egg-shell consists mainly of carbonate of lime, and when freshly laid is covered by a mucous coating. The egg-white consists of 86.2 per cent. of water, 12.3 per cent. nitrogenous matter, 0.2 per cent. fat, and 0.06 per cent. ash. The yolk consists of 49.5 per cent. water, 15.7 per cent. nitrogenous matter, 33.3 per cent. fat, and 1.1 per cent. ash.[1] These are averages; different eggs vary somewhat in composition from each other. It is noteworthy that eggs contain practically no carbohydrates.

In addition to fresh and refrigerated, eggs are classified in the trade as "rots," "spots," "checks," "ringers," "chickens," "dirty shells," "heated," or "incubated," etc. Eggs are assorted by inspection and candling. Candling consists in holding them before a bright light; the egg is translucent and the movable yolk may clearly be discerned, as well as the air space which is always at the larger end. A practiced eye quickly detects eggs that are not first quality. Rotten eggs are distinguished as "red rots" and "black rots," depending upon the kind of putrefaction. By "spots" are understood eggs that contain opaque spots under the light. These spots usually consist of local growths of mold that have penetrated a crack in the shell, although they may be due to coccidia, embryos, or foreign bodies. "Checked" eggs are those which have slight cracks or nicks in the shell. "Ringers" contain small embryos of about two days' growth, which are flat, disk-like, and reddish in appearance. "Chickens" contain embryos of larger growth. Eggs with dirty shells are undesirable more from esthetic than other reasons. The dirt usually consists of hen excrement. A "heated" egg is a shrunken egg, that is, an egg that has been exposed to the summer temperature for several days. Some water is lost by evaporation through the porous shell, the air sac on the end has increased considerably in volume, and in many instances the embryo is partly developed; therefore, heated eggs are also known as incubated eggs. Many of the eggs gathered during the hot months of summer, especially in July and August, belong to this category. These eggs are much less desirable than the spring and fall layings. Eggs are also graded as to size, the very small eggs being undesirable, commanding a lower figure in the market. Further,

[1] Pennington: "A Chemical and Bacteriological Study of Fresh Eggs," *Jour. Biol. Chem.*, Vol. VII, No. 2, Jan., 1910, p. 110.

eggs are classified as strong- or weak-bodied, depending upon how they "stand up" when broken out.

Eggs as they come from the hen frequently contain bacteria, worms, gravel, blood clots, and foreign bodies of various kinds. Practically all eggs contain bacteria, although numerous observers report occasionally that an egg is sterile. As a rule, these observations are based upon planting a small part of the egg. If the entire egg is planted a growth is almost invariably obtained. Thus, in eighteen freshly laid eggs which I recently examined every one of them contained bacteria in the yolk; two of them contained *B. coli.* Curiously enough, there are practically always more bacteria in the yolk than in the white; the white contains some bactericidal property, probably similar to that possessed by fresh blood. The bacteria doubtless gain entrance to the egg while in the oviduct. Pernot [1] examined the eggs from over the size of a pea to the perfect egg and found bacteria at every stage. It is well known that the bacteria may also get into an egg through the shell, as it is porous and permeable. When the shell is moist and dirty the chances of growth and mold piercing it are increased. Eggs laid in the summer time (July and August) contain many more bacteria than those laid in the spring, fall, and colder months. It is well known that summer eggs do not keep as well as winter and spring eggs.

Very large quantities of eggs are now broken out, mixed, frozen, or dried. These products are largely used by bakers and others who use eggs in quantities.

Of all foods, so far as known, eggs are less liable to convey disease or contain harmful properties than any other single food of animal origin. The literature is singularly free of instances of sickness attributable to eggs. There is no known infection of the hen transmissible to man through its egg. Eggs do not agree with some people, who have an "idiosyncrasy," so that a very small quantity will bring on symptoms resembling anaphylaxis. This condition is doubtless an instance of specific hypersusceptibility to egg protein. There are several cases on record in which this hypersusceptibility has been cured by the administration of pills or candy containing at first infinitesimal amounts of egg-white, gradually increasing the amount. The entire treatment should extend over a period of months. In this way an immunity may be established in man precisely analogous to the immunity which may be established by repeated injections of an alien protein into guinea pigs.

[1] "Investigation of the Mortality of Incubator Chicks," *Bull. 103,* Oregon Agr. College Exp. Station.

CHAPTER IV

PLANT FOODS

Vegetable substances may be injurious to health from several causes. Many plants contain a physiological poison, such, for example, as aconite, strychnin, ricin, abrin, muscarin, and a long list of other substances normally present. Certain plants develop poisons, such as rye, which causes ergotism; spoiled corn, responsible for pellagra; polished rice, associated with beri-beri; the chickpea, responsible for vetch poison or lathyrism. The plant may be entirely wholesome when fresh, but may develop poisons as the result of bacterial action. Changes occur in vegetables entirely similar to those which occur in nitrogenous animal products, with the production of poisons of different kinds. The same microorganisms that produce "ptomains" or other toxic substances in meat when introduced into vegetables act in the same manner. The richer the vegetable in nitrogen the more likely is it to acquire such poisonous properties. Carbohydrates retard or suppress this action; therefore, vegetables containing large amounts of carbohydrates are less apt to become dangerous.

Certain vegetables, such as lettuce, celery, radishes, and similar plants, eaten raw may convey typhoid fever, cholera, dysentery, both amebic and bacillary, the eggs and larvæ of animal parasites, and other infections.

All vegetables which are eaten raw should be washed thoroughly beforehand, otherwise they may be contaminated with manure and other impurities or the excrement of domestic animals which have been roaming in the garden. The larvæ of tapeworms and roundworms have been transmitted to man in this manner. Water from foul wells is used sometimes for sprinkling gardens, and it is possible for typhoid and other intestinal infections to be spread by this means when the vegetables are eaten raw.

POISONING FROM PLANT FOODS

Ergotism.—Ergotism is a form of food poisoning brought on by prolonged use of meal made from grain contaminated with the *Clavi-*

ceps purpurea. The fungus develops in the flowers of rye and other grains. The chief source of the poisoning in man is from rye, in which case the fungus may entirely replace the grain. Ergotism is practically unknown in this country, but in Europe it is still occasionally met with, although not to the same extent as in former times. From ergot Kobert was able to isolate three poisonous substances, sphacelinic acid, cornutin, and ergotin. Sphacelinic acid is a non-nitrogenous, unstable body and is believed to be the active agent in contracting the blood vessels. Cornutin is also an active alkaloid and produces vasomotor contraction. According to Novy, more recent investigations have made it probable that there are other substances present which constitute the real toxic agent. Thus, Jacoby obtained a non-nitrogenous resin sphacelotoxin which he regards as the specific poison. The intoxication may have an acute or chronic course, and in either type the symptoms may be nervous or convulsive, or else trophic or gangrenous in character.

The presence of the sclerotium may be suspected from the color of the meal, which is grayer than usual and often shows violet-colored specks. The addition of potassium hydroxid with heat produces an odor of trimethylamin resulting from the breaking up of the grain containing chinolin. Further, the grain contains a dye which is soluble in alcohol or ether. To 10 grams of the meal add 10 c. c. of ether and 20 drops of dilute sulphuric acid. Shake well and filter after half an hour. Then add several drops of a saturated solution of sodium bicarbonate, which dissolves out all the coloring matter.

Lathyrism.—Lathyrism or vetch poisoning is a rather rare condition met with in some parts of Europe, notably Austria and Italy, in northern Africa, and in India. The vetch seed is ground in the form of meal and used as a partial substitute for that of wheat. The seed is popularly known as chick-pea. The vetch seeds are obtained chiefly from *Lathyrus sativus* and *Lathyrus cicera.* The eating of bread prepared from meal containing the seeds of the lathyrus is followed by sudden and severe pains in the lumbar region, girdle sensation, motor paralysis of the lower extremities, tremor, and fever. The nature of the poison is not known, but it is probably of the nature of a toxalbumose, of which ricin and abrin, the poisons of the castor bean and the jequirity bean respectively, are well-known examples.

Mushroom Poisoning.—The ill effects from eating mushrooms are due to mistaking the poisonous for the edible species. The number of species of poisonous mushrooms which are capable of causing death is not very great. The *Amanitas* and the *Volvarias* are almost exclusively the poisonous species. The following is a list of the most poisonous mushrooms known, and all that are likely at any time to produce death:

Amanita phalloides Fr.
Amanita citrina Pers.
Amanita verna Bull.
Amanita virosa Fr.
Volvaria gloiocephala, var. speciosa (Fr.).
Amanita muscaria (L.) Pers.
Amanita pantherina DC.
Lactarius torminosus (Schaeff.) Fr.
Lactarius rufus Fr.
Lactarius zonarius (Bull.) Fr.
Lactarius pyrogallus (Bull.) Fr.
Russula emetica Fr.
Russula queletii Fr.
Russula fœtens (Pers.) Fr.
Boletus felleus Bull.
Boletus satanus Lenz.
Boletus erythropus Cke.
Boletus luridus Schaeff.
Entoloma lividum Bull.

The *Amanita phalloides,* or fly fungus, is an exceedingly poisonous, dangerous, and seductive species, responsible for most of the deaths from toadstool eating. Because of its white color it is mistaken for the common mushroom, *Agaricus campester.* The *Amanita phalloides* does not grow in the woods, neither has it white gills nor white spores nor a volva at the base of the stem. The poisonous principle is a syrupy alkaloid known as muscarin. The alkaloid is without taste or color. It produces powerful intoxicating effects somewhat analogous to pilocarpin in its action and antagonized by atropin. The alkaloid is soluble in water, and mushroom poisoning may be prevented by soaking the mushrooms in water slightly acidulated with vinegar before they are cooked.

Potato Poisoning.—It has long been known that potatoes contain normally a very small amount (about 0.06 per cent.) of the poisonous principle *solanin,* but it is only quite recently that it has been discovered that, under certain circumstances, they may contain the poison in amounts sufficient to cause grave disturbance of the system. The increase is due to the action of at least two species of bacteria, *Bacterium solaniferum non-colorabile* and *Bacterium solaniferum colorabile,* and occurs in those tubers which, during growth, have lain partially exposed above ground, and in those which, during storage, have become well sprouted. The most extensive outbreak of potato poisoning recorded occurred in 1899 in a German regiment, fifty-six members of which, after eating sprouted potatoes, were seized with chills, fever, headache,

vomiting, diarrhea, colic, and great prostration. Many were jaundiced and several collapsed, but all recovered. Samples of the remaining potatoes yielded 0.38 per cent. of solanin, and this would indicate that a full portion must have contained about five grains (Osler).

Beri-beri.—Our knowledge of beri-beri is now sufficient to place this scourge of the tropics among the preventable diseases. Long associated with rice, it is now evident that beri-beri is a disease due to an unbalanced or monotonous diet made up largely of polished rice, that is, rice without the pericarp. The disease may be prevented or cured by the administration of rice bran.

Beri-beri, or kakke, is a specific form of multiple peripheral neuritis occurring endemically, or as an epidemic, in most tropical or subtropical climates. It is characterized clinically by disturbances of motion, sensation, dropsy, and affection of the heart. The symptoms are attributable to degenerative changes in many of the peripheral nerves, being a toxic neuritis similar in many respects to that produced by alcohol, arsenic, and other metals, or the toxone of diphtheria. Three types of the disease are recognized: (1) the paraplegic, or dry; (2) the dropsical, or wet; and (3) the mixed. The course of the disease is uncertain; sudden death owing to involvement of the heart is a common termination. Recovery is frequent and may be complete; it is promoted by change of climate, improvement in the sanitary surroundings, and especially by change of diet. Two main views as to the nature of the disease have long prevailed: (1) that it is an infection, (2) that it is a food poisoning.

Beri-beri has some of the earmarks of an infectious disease, resembling malaria somewhat, so far as telluric influences and seasonal prevalence are concerned. Scheube insists that beri-beri is an infectious disease because: (1) young, strong, and well-nourished persons are most frequently attacked, and particularly liable to the severest form of the disease; (2) beri-beri has not only its definite geographical region of distribution, but even in beri-beri countries it does not occur everywhere, being confined to certain narrow, sharply limited districts; (3) the maximum of frequency of the disease is during that season which is, first of all, distinguished by great moisture, and, secondly, by a high temperature liable to many variations;[1] (4) during recent decades beri-beri has obtained a considerable distribution in certain countries, as Java, Japan, and Brazil, without any change of food having taken place among the people. The nature of the virus is not known, and the various microorganisms that have been described in the blood and tissues are in all likelihood not the specific excitants.

Takaki, the surgeon-general of the Japanese navy, believed that beri-beri is due to nitrogen starvation. He has practically abolished

[1] Hirsch.

beri-beri in that service by allowing a larger portion of nitrogenous food and forbidding the use of fresh fish altogether. It must be recalled, however, that this dietetic change was coincident with other sanitary reforms. In former years one-fourth of the personnel of the Japanese navy suffered from beri-beri; now it is almost unknown.

Manson's theory of beri-beri is interesting. He believed that it is undoubtedly a germ disease, but that it is produced by a harmless saprophyte which develops a poison in food outside of the body.

Many physicians who have studied the subject in Japan, Java, the Philippines, and other countries have long regarded rice as the important cause of the disease. In the prisons of Java the proportion of cases is 1-39 when rice is eaten completely shelled, 1-10,000 when the grain is eaten with its pericarp. In some places the disease has disappeared when the unshelled rice has been substituted for the shelled.

Eykman first pointed out that beri-beri is not attributable to rice in general, but that certain kinds of rice prepared in certain ways are more liable to produce the disease. It is now known that a disease resembling beri-beri characterized by degeneration of the peripheral nerves may be produced in fowl by feeding them on white or polished rice. Furthermore, the same changes in diet which avoid or cure beri-beri in man act in a similar manner in respect to this polyneuritis in fowl. It has now been established that polished rice causes beri-beri if the diet is based almost exclusively on this foodstuff, but that, if a sufficient amount of other things, such as fresh meat and vegetables, are taken with it, the disease is not produced. In the polishing of rice the pericarp or cortical portion of the grain is removed and the embryo is discarded. It is evident that these discarded portions contain some substance essential to a well-balanced ration. It has been found that most of the phosphorus is contained in the pericarp. The amount of phosphorus is a good guide in the selection of a beri-beri-preventing rice. In the East rice is regarded as unsafe if it contains upon analysis a content of less than 0.35 per cent. of phosphorus pentoxid. It is not, however, the absence of the phosphorus which induces beri-beri, but the amount of phosphorous, as phosphorous pentoxid (P_2O_5) may be taken as an index of the degree to which the rice has been polished. The particular substance responsible has not as yet been isolated.

The recent work of Fraser and Aron, Breaudat and Denier, Dehaan, Heiser, and others leaves little doubt concerning the relation of polished rice to beri-beri. Heiser[1] reports that, prior to February, 1910, polished rice was commonly used in the Culion leper colony. The deaths from all cases between February, 1909, to 1910 were 898, of which 309 were due to beri-beri. From February, 1910, to February, 1911, un-

[1] *Jour. A. M. A.*, Vol. LI, 1911, p. 1237.

polished rice was used, and there were 369 deaths, a reduction of over one-half the death rate for the previous year. It is significant that there were no deaths from beri-beri during this interval following the use of unpolished rice. Heiser further reports 50 cases of beri-beri treated by giving daily 15 grams of rice polishings. Improvement was noticed in all except two very advanced cases. These results have been so striking that the Philippine government has drafted a bill providing for the general use of unpolished rice; that is, rice containing at least 0.4 per cent. of phosphorus as phosphorus pentoxid, and the levying of a tax upon polished rice which makes its sale practically prohibitive. Breaudat and Denier[1] at Saigon, in Indo China, report good results from the prophylactic use of rice bran. Forty grams are administered daily in the ordinary food. No case of beri-beri developed among 49 native soldiers who took bran, while 17.4 per cent. of 311 controls developed the disease.

The prevention of beri-beri in the Philippine Islands based upon the rice theory is little short of marvelous. The disease has been entirely eliminated from the Philippine native scouts owing to the reduction in the amount of rice from 20 to 16 ounces, a substitution of undermilled rice for the polished article, and the addition of a legume to the dietary. In 1908 and 1909 there were 600 cases of beri-beri annually. In the entire 17 months since the alteration in the ration went into effect there have been but 7 cases of the disease; occasionally cases may be expected owing to disobedience of instructions.

Infantile beri-beri is also common in the Philippines, and may likewise be prevented and even cured with rice bran.

Prevention.—The prevention of beri-beri consists in substituting the use of whole rice for the polished grain; also in improving the quality of the food and in providing for better balanced dietaries. The prophylactic value of rice bran added to the ordinary diet must be borne in mind.

It should be borne in mind that beri-beri may be produced by a monotonous diet of other starchy substances, such as wheat flour (Little and Strong). A varied diet is, therefore, one of the prime essentials in the prevention of beri-beri. There are certain accessory factors favoring beri-beri which must be taken into account. The disease occurs especially in overcrowded places, such as ships, jails, and asylums; during the hot and moist seasons; and following exposure to wet. These are to be avoided. Europeans living under good hygienic conditions, and enjoying a well-balanced diet, rarely contract the disease.

Manson advises that when beri-beri breaks out in a school, jail, or similar institution the place should be emptied of its inmates as soon as possible; at all events, those parts of the building in which the dis-

[1] *Ann. de l'Inst. Pasteur,* Feb., 1911, No. 2.

ease first appeared should be cleared out, and not reoccupied until they have been thoroughly cleansed, disinfected, ventilated, and dried. Overcrowding must be strictly avoided; ventilation must be effective. The dietary should be revised and, if necessary, rice eliminated from it as much as possible. In the place of rice fresh meat, vegetables, and wheat flour should be substituted. All the inmates should be obliged to pass the largest part of every day in the open air. Their knee-jerks should be tested and their legs examined for numbness, edema, and muscular hyperesthesia from time to time. Any suspicious cases should be removed and treated at once.

Pellagra.—For the present pellagra is included among the diseases due to poisonous food, for the bulk of evidence indicates that it is caused by spoiled corn. This disease is in all probability another example of a food intoxication caused by some toxicogenic saprophyte. There is also a suspicion that insects (*Simulium* or *Stomoxys*) may be concerned in its transmission. Pellagra is largely a preventable disease in which the social conditions loom large; it is especially prevalent where poverty, overcrowding, and misery prevail. It occurs both sporadically and endemically.

The history of pellagra in the United States is interesting, especially in view of the fact that corn is indigenous to America and is extensively used by large portions of the population, especially in the South. Nevertheless, compared to its ravages in Italy, Roumania, and other countries, we have escaped pellagra as a scourge. This is due perhaps to the fact that, owing to climatic conditions, the corn here is permitted to mature before it is garnered and is, therefore, less apt to spoil. However, during the past decade or two the corn belt has gradually been pushed farther and farther north. This means that it is often harvested before it is mature, and the chances of its spoiling are favored in transporting it to our southland in a moist condition. A carload of corn starting from the Great Lakes may ferment and become so overheated on its journey south that occasionally it catches fire spontaneously. These facts may account for the notable increase in the amount of pellagra in our southern cities. The disease was first recognized in America 46 years ago (1864) by Dr. Gray, of Utica, New York, and by Dr. Tyler, of Somerville, Mass., who each reported a case of probable pellagra. It was overlooked until 1906-1907, when Searcy reported an epidemic in the Alabama Insane Asylum. In the same year (1907) Babcock's article on the cases in the State Insane Asylum of Columbia, South Carolina, aroused our present revival of interest in the disease. In 1908 Wood and Lavender found four cases in Wilmington, North Carolina. Since then a flood of cases have come to light all over the country, especially in the south; outbreaks, however, occur as far north as Peoria, Illinois, where 40 to 50 well-marked cases out of 2,200 inmates were discovered in the State Hospital for the Insane.

Lavender now estimates that there are between 25,000 and 50,000 pellagrins in the United States.

The disease appeared in Italy about 1750, but was first described there in 1771 by Frapolli, of Milan, who applied the name "pellagra" (Italian *pelle,* skin, and *agra,* rough). Marzari in 1810 first called attention to the relation between maize and pellagra. In 1844 Balardini suggested the theory that the disease might be due to spoiled maize, that is, maize which had undergone fermentative change by reason of the growth of fungi on the grain. At present pellagra is most prevalent in northern and central Italy and in Roumania. Triller states that in 1906 there were 30,000 pellagrins in Roumania; in certain parts of Italy as much as 30 to 50 per cent. of the population have the disease; in 1899 there were nearly 73,000 sick with the disease in all Italy, this being upward of 10 per thousand of the rural population. The disease also occurs in Spain, Corfu, Asia Minor, Austria, Servia, Bulgaria, and occasionally in India, Africa, Barbadoes, Mexico, South America, and Egypt.

As preventive measures must be based entirely upon our conception of the etiology of the disease, it is necessary to consider briefly some of the views upon this subject. It is the accepted opinion of most students of the disease that pellagra is an intoxication due to using Indian corn (maize) as a food, which, under the influence of some parasitic growth (bacteria or fungus), has undergone certain changes with a production of one or more toxic substances. Lombroso, who studied this subject for years, made alcoholic and watery extracts from spoiled maize and obtained chemical substances of an undetermined nature, which were given to men and animals with the production of symptoms analogous to pellagra. The interpretation of all such work is as yet, however, in an uncertain state.

With regard to the parasites found on maize, it may be said that the varieties are numerous, and no single one seems to be constant enough to be rated as the definite causative agent. Seni incriminates the *Aspergillus fumigatus* as the cause of the maniacal form of pellagra, and the *Aspergillus flavescens* as the cause of the depressive form. These molds have resisting spores which withstand heat, hence ordinary cooking is not sufficient to destroy them. The *Bacterium maydis* has also been associated with the disease. Lombroso, as a result of his studies, maintained that pellagra is due to a poison (toxine) developed in maize by microorganisms (molds or bacteria), in themselves harmless to man (that is, saprophytes).

Other views concerning the nature of pellagra are: that it is an autointoxication, the poisonous substances being produced in the bowels as a result of the constant and almost exclusive diet of corn, which produces certain changes in the intestinal flora, and the production of poisonous

substances. A somewhat similar view is that the disease is an intestinal mycosis, the offending microorganisms being eaten with corn and colonizing in the intestinal tract. Others regard the disease as of an infectious nature, and several parasites have been reported in the blood and organs. In France especially the idea has been brought forward that pellagra is not a definite morbid entity at all, but a symptom-complex sometimes observed in alcoholics and cachectic states of diverse origin, the erythema being regarded only as a common solar erythema. Sambon, as the result of epidemiological studies, brought forward (1905 and again recently) the view that pellagra is an insect-borne disease, and incriminates the *Simulium reptans.*

Raubitschek[1] recently brings forward evidence that pellagra depends upon some noxious substance (*noxe*) activated by the action of sunlight. This is the photodynamic theory, and corresponds to the action of light upon a photographic negative. It is suggestive that the skin lesions in pellagra are mainly confined to the exposed surfaces. There is also a substance in buckwheat poisoning (*fugopyrismus*) that affects animals exposed to the light, but not those kept in the dark.

Pellagra usually runs a chronic course, with acute exacerbations, which usually occur in the spring and fall of the year. The disease sometimes runs an acute and rapidly fatal course. The development seems to be more rapid and grave in children. The poison, whatever its nature, produces toxic and trophic manifestations. The triad of symptoms are: (1) digestive disturbances, (2) erythema, and (3) nervous disturbances. The final scene usually includes profound cachexia, great muscular weakness, and insanity.

Corn.—Maize or Indian corn is a native of the Western Hemisphere and was cultivated by most of the northern and western tribes of North American Indians before Columbus reached these shores. The importance of the corn crop to-day may be gathered from the fact that, according to the census of 1900, almost one-third of all the land under cultivation in the United States was devoted to corn. It was grown on 88.6 per cent. of all the farms in the country in the crop for 1889. The value of the annual crop now exceeds a billion dollars. Corn contains 24.7 per cent. of water. The water-free material consists of 12.7 per cent. proteins, 4.3 per cent. fat, 79.3 per cent. starch, sugar, etc., 2 per cent. crude fiber, and 1.7 per cent. of mineral matters. The several nutrient substances in corn and other common cereals are much the same; the individual compounds, however, making up these groups differ considerably.

The kernel or seed, it must be remembered, is not inert, but a living thing which, under favorable conditions, will develop into a new plant, and each part of it is made up of cells especially fitted for a particu-

[1] *Berliner klin. Wochens.*, Vol. XXIII, No. 26, June, 1910.

lar rôle in this process of reproduction. Roughly speaking, a seed consists of three divisions: the skin, the germ, and the endosperm. It is a well-known fact that corn, when allowed to ripen before it is taken from the stalk, keeps much better than immature corn. It is certain that protective substances (antibodies) are developed in the kernel which retard the growth of bacteria and molds. Moist corn kept warm spoils readily, whereas corn once thoroughly dried is proof against serious fermentative changes.

The tests for spoiled corn are not entirely satisfactory. They may be divided into physical, biological, and chemical tests. The physical test consists mainly in the luster, the absence of molds, the odor, and taste. The biological test consists in planting the corn; from 90 to 95 per cent. should germinate. The chemical test includes among other determinations the proportion of ash after burning, and Gosio's phenolic reaction with ferric chlorid. A green purple color with this reagent indicates fermentation, with the production of phenolic compounds.

Spoiled corn may be renovated by polishing and then heating, to prevent further growth of molds. It is difficult to detect renovated corn by inspection alone, but the biological test will disclose whether or not it has been heated. The practice of renovating corn should either be prohibited or be placed under strict official control.

There are three hundred or more varieties of corn. It is quite practicable to raise corn either with a high protein content or a high fat content. Smith, at the Illinois Agricultural Experiment Station, in ten generations raised the oil or fat content from a minimum of 4.7 per cent. to a maximum of 7.337 per cent. Such corn is sought after for the fattening of hogs. While of advantage to hogs, it may be detrimental to man, for when corn becomes moldy it is always the embryo that is affected first, and there the fungus flourishes best. According to Alsberg, the greater part of the toxic material is in the decayed embryo. It happens that the greater part of the oil is also located in the embryo. The variety rich in oil is probably also one with a large germ. It is, therefore, possible that corn with a large embryo and high fat content may spoil more readily and produce a greater amount of the poison responsible for pellagra.

It should be borne in mind that, while corn itself may not be the direct cause of pellagra, it may hold the same relation to that disease that the swamps bear to malaria.

Prevention.—The line along which pellagra prophylaxis is planned depends entirely upon our conception of the disease. As pellagra prevails among the poor, especially those who live under uncleanly and squalid conditions, it at once becomes evident that economic and social improvements are an important part of the program. Prophylaxis spells prosperity in this disease as in others. The Italian struggle culminated

in the law of 1902 for "the prevention and cure of pellagra," which was inspired by Lombroso's views upon the disease. The Italian measures may be summarized as follows: those aimed at the cure of the disease are a free distribution of salt (a government monopoly in Italy), the distribution of food either at the homes of the patients or through sanitary stations, and the treatment of severe cases in hospitals for pellagrins and in insane asylums. The prophylactic measures are mainly directed against the use of spoiled corn as an article of food. They comprise a census of the disease and a report of all cases; the testing of corn and meal brought in at the frontiers or offered for sale to the mills and the prohibition of its sale if found spoiled; the exchange of good corn for spoiled corn; desiccating plants; cheap coöperative kitchens; the improvement of agriculture; and the education of the people. The corn is inspected by experts and is submitted to certain tests. If found spoiled, its sale for food is prohibited. The tests are not entirely satisfactory from a scientific standpoint, but seem sufficient for practical purposes. According to Mr. Cutting, the weak point in the inspection of corn seems to be in dealing with home-grown corn, especially the meal, either at the mills or on the markets. There seems to be no solution of this difficulty except governmental ownership of the mills. The agricultural improvements are directed toward teaching the use of better varieties of corn and proper methods of culture, handling, etc., or how to supplant corn entirely with a more profitable crop.

The desiccating plants for the artificial drying of corn are considered a very important prophylactic measure, as they prevent the spoiling of the grain. These desiccators are of two types, fixed and portable, and there are a large number of public desiccators throughout Italy. There is also a provision in the law for public storehouses properly constructed, where the grain may be stored under the best conditions to prevent spoiling. Rural bakeries and economic kitchens are establishments where an effort is made to eliminate from the peasant's diet bread made of corn, by supplying good white bread and other food at a low cost. Above all, however, stands the education of the people to the dangers of spoiled corn, and the healthfulness of a varied diet and better living conditions.

The results of the campaign in Italy seem to be a diminution in the amount of the disease in central Italy. Strange as it may seem, however, the disease is increasing its area, and parts of Italy previously free from pellagra are now developing the disease.

SECTION IV

AIR

CHAPTER I

COMPOSITION OF THE AIR

The air constitutes a gaseous ocean in which we live; it consists of a vast volume of gases at least one hundred miles high.[1] Ordinarily we speak of this gaseous envelope of the earth as the atmosphere, and the water resting upon the surface of the earth as the aquasphere, while the solid structure of the earth is called the petrosphere. Between the atmosphere on one hand and the petrosphere and aquasphere on the other hand is the region of most abundant life, and this is spoken of as the vivosphere.

An abundant supply of fresh air is necessary at all times. The importance of fresh air was almost completely ignored in practical life until recently—thanks to the tuberculosis propaganda. While recent studies have shown that the air is not to be feared as a frequent medium for conveying specific infections, it has been demonstrated that an abundant supply of fresh air is necessary to perfect well-being. Statistical studies seem to prove that, of the predisposing causes of sickness which are usually in action, impurities of the air are perhaps the most important. This has been demonstrated over and over again in the case of horses, cattle, and dogs, as well as in men confined in badly ventilated barracks, jails, and other places.

Many other factors are now known to be a greater menace to health than the "bad" air of crowded places; sanitarians, however, have come to regard an abundant supply of pure fresh air, well conditioned, as one of the real essentials for health and maximum efficiency.

Further, it should be remembered that the combustion of the food we eat depends upon the oxygen of the air we breathe, and that digestion and metabolism are stimulated and improved by an abundant supply of fresh air or rendered sluggish and retarded by prolonged exposure to vitiated air.

[1] Forty-five or fifty miles is its practical limit, and anything beyond that distance is in an extremely tenuous state.

The atmosphere is now known to contain the following gases in the following approximate proportions by volume, measured at 0° C. and at 760 mm. pressure:

	Volumes per Cent.	Weight per Cent.
Oxygen	20.94	23.2
Nitrogen	78.09	76.9
Carbon dioxid	0.03	
Argon	0.94	
Helium, krypton, neon, xenon, hydrogen, hydrogen peroxid, ammonia, ozone	traces	

"Pure" air, in addition, contains water vapor in varying amounts, dust, radioactive substances, etc.

The air is a mixture of gases and not a chemical compound. The proofs of this are manifold: (1) the gases do not exist in the air in the proportion of their combining weights or any multiple of them; (2) on mixing the gases in atmospheric proportions there is no heat evolved; (3) the composition of air within limits is variable; (4) when water dissolves air it dissolves each gas according to its partial pressure and its own proper coefficient of solubility. Thus, air contains more nitrogen than oxygen, but, oxygen being more soluble, water takes up 1.87 parts of oxygen to 1 part of nitrogen.

It was Jean Mayow in 1669 who first proved that air was not an element, but a mixture of gases, and later Lavoisier discovered the two gases which about 100 years later were separated by Priestley and Sheele.

The composition of the air shows wonderful uniformity all over the earth's surface wherever examined. This is due to the enormous amount of atmosphere and the mixing influences of air currents. However, in confined spaces where the air is not in motion, especially where decomposition of organic matter is taking place or where active combustion is going on, or in the presence of animal life, the composition of the air varies considerably.

The difference in composition between inspired and expired air is as follows:

	O	N	CO_2
Inspired air	20.81	79.15	.03
Expired air	16.033	79.557	4.38

The expired air is also warmer, is increased in volume, and contains more moisture, but fewer particles, such as dust and bacteria. Under normal conditions of quiet respiration the expired breath contains no bacteria.

OXYGEN

About one-fifth (20.94 per cent. by volume, 23.2 per cent. by weight) of the atmosphere consists of oxygen, which in many respects is its most important element. Slight differences are noted; thus, the air of towns contains somewhat less (20.87 per cent. by volume) than in mid-ocean. The slight differences that have been noted in the percentage of oxygen are of no special importance. It may drop to 13 per cent. or may rise to 50 per cent. or even higher without any very apparent alteration in the vital functions. An atmosphere containing only 11 to 12 per cent. of oxygen becomes dangerous, and 7.2 per cent. results in death. The constant percentage of oxygen is due in part to the enormous amount of it. Flügge estimates that at the present rate at which the oxygen is used by respiration and combustion it would take eighteen thousand years to reduce it by one per cent., even if not replaced by vegetation. The lungs, of course, at no time after the first breath contain air with the full percentage of oxygen. This is owing to the fact that the lungs do not completely empty themselves, and the residual air remaining in the lungs accumulates carbon dioxid and loses oxygen.

Oxygen is the element in the air that sustains all life. It is absorbed by the lungs, passes into the blood, combines loosely with the hemoglobin of the red blood corpuscles, and is thus carried to all the tissues and cells of the body. Oxygen in combination with the hemoglobin forms an unstable compound—oxyhemoglobin—which gives the bright red color to arterial blood. The oxygen bound with the hemoglobin in arterial blood consists of from 22 to 25 per cent. of the volume of the blood. The amount of oxygen absorbed varies with the age, condition of health, and activity. According to Professor Foster, the average person inhales in 24 hours about 34 pounds of air, which corresponds to a little over 7 pounds of oxygen. As the lungs absorb about one-fourth of the oxygen inhaled, it appears that the average amount of oxygen absorbed daily is nearly two pounds. Oxygen also exists in its gaseous form in blood, saliva, bile, urine, and other fluids of the body, but only in minute amounts.

The amount of oxygen in the air may readily be measured in the Petterson-Palmquist or Haldane apparatus. The oxygen is absorbed by 10 per cent. oxalic acid in a saturated solution of KOH (sp. gr. 1.058); the difference in volume before and after absorption represents the amount of oxygen (pp. 591-593).

Determinations of the amount of oxygen of the atmosphere have no particular hygienic importance.

NITROGEN

The nitrogen in the air may be regarded as a diluent, so far as its direct action upon man is concerned. There is no appreciable difference in the amount of nitrogen contained in inspired and expired air. Although inert, it is very important, for it serves to dilute the oxygen and thus regulate the rate of combustion and its prototype respiration. The nitrogen is of more direct importance to plants, as some are able to fix a certain amount of atmospheric nitrogen through the action of certain bacteria, as *B. radicicola,* in the root nodules. While nitrogen in the atmosphere seems to be an indifferent element and has no hygienic significance, it is a constant constituent of all protein matter. The amount of nitrogen dissolved as a gas in the blood and body juices increases proportionately with the pressure (P. Bert).

ARGON

Argon, discovered in 1894 by Lord Rayleigh and Professor Ramsey, is quite inert chemically; that is, it has not been made to combine with any other element. It comprises from 0.75 to 1 per cent. of the atmosphere. Argon has not been demonstrated in the body; it is apparently indifferent, and, so far as our present knowledge goes, has no hygienic significance.

OZONE

Ozone, described by Schönbein in 1840, is rarely found in the air in greater proportions than mere traces, but it is so potent chemically that even small quantities may be of importance. At Montsouris, after years of observation, the largest quantity of ozone found in outside air was 1 part in 700,000. Ozone may be regarded as a normal constituent, though by no means constant in air. It is generally absent in the air of large towns and cities, and almost never present in the air of inhabited rooms. It is most abundant at sea and near woods.

Atmospheric ozone is formed in nature during electric discharges, by the oxidation of phosphorescent substances; and perhaps by the respiration of plants; also by friction of large masses of water, such as the sea against the air.

Ozone consists of three atoms of oxygen instead of two, compressed into a molecule, thus: $3O_2=2O_3$. It is one of the most powerful oxidizing agents known, and in small amounts is exceedingly irritating; in large amounts it is fatal to life. Ozone is one of our most active bleaching agents, and in proper concentration is one of the most potent

germicides known, and has been used to sterilize water, to disinfect bandages, and for other purposes.

The direct effect upon health of the minute traces of ozone ordinarily found in the atmosphere must be very small. From what we know of its properties, it would at once combine with the organic matter in the nostrils and upper respiratory passages. The presence of ozone in the air indicates the absence of organic impurities. That is one reason it is not ordinarily found in the atmosphere of an inhabited room.

It requires at least 13 parts of ozone per million in the atmosphere to influence bacteria. Such large proportions are never present under natural conditions. Comparatively small amounts are irritating to the respiratory mucous membrane. Thus, Hill and Flack[1] have studied the action of pure ozone (free of contaminating oxids of nitrogen), and find it irritating in the proportion of one part per million. Exposure for two hours to a concentration of 15 to 20 parts per million endangers life. Hill and Flack conclude that there is no harm in breathing weak concentrations of ozone, such as can scarcely be perceived by a keen sense of smell.

Recently ozonizers have been placed upon the market for the purpose of purifying the air of rooms; these are poor substitutes for ventilation. Not only may the ozone itself be harmful, but the higher oxids of nitrogen may be formed when the electric current acts upon moist air.

The *tests* for ozone depend upon the fact that it oxidizes the color of tincture of guaiac, causing it to turn blue. It also acts upon potassium iodid, and turns starch in presence of free iodin a blue color: $2KI+H_2O+O=2KOH+I_2$.

HYDROGEN PEROXID (H_2O_2)

Hydrogen peroxid may be found in appreciable traces in rain and snow. One liter of rain or snow water contains about 0.182 mg. of hydrogen peroxid. This higher oxid gives many of the reactions of ozone, being a very active oxidizing agent, and care must be exercised not to confuse them.

AMMONIA

The ammonia in the air comes largely from the decomposition of organic matter. It is produced in sufficient quantities in a manure heap to be perceptible to the senses. Ammonia may be regarded as one of the normal constituents of the atmosphere, as it is constantly pres-

[1] *Proceed. Royal Society*, London, B, 1911, LXXXIV, 404.

ent in slight traces; it varies in distribution, more being found in the lower stratum of air near the soil. It exists both in the free state and also combined as nitrate and carbonate. Daily analysis of the air at the observatory at Montsouris for five years gave, as a mean for ammonia, 2.2 mg. per 100 cu. m. There is less after rain, because it is absorbed by the water during its passage through the atmosphere.

Albuminoid ammonia, according to Angus Smith, is a measure of the sewage of the air; that is, the amount of organic impurities, both living and dead.

MINERAL ACIDS

The atmosphere at times contains nitric, sulphuric, and other acids. These are derived from electric discharges, but mainly from the combustion of coal and from industrial processes. Sulphuric acid or sulphates in the air, according to Angus Smith, is a measure of manufacturing activity and also of decomposition. In other words, it is part of the oxidized and, therefore, purified sewage of the air. Traces of sulphuric and sulphurous acids exist in the air. The sulphates and sulphites are usually present as ammonia salts. These substances are usually present in such small amounts that they are appreciable only when washed into rain or snow. A liter of rain water may contain from 0.7 to 2.99 mg. of sulphuric acid. More of this acid is found in the air about industrial centers than in the air over country or sea. The sulphuric acid in the air comes mainly from the sulphur in coal.

CARBON DIOXID

Carbon dioxid (CO_2) is a very important constituent of the atmosphere. The amount of this gas in the air is relatively small—normally about 0.03 per cent., usually expressed as 3 parts in 10,000. When we consider the great bulk of the atmosphere the total amount of carbon dioxid is very great. It is estimated that there is more carbon in the form of carbon dioxid in the air than there is in all other forms on the earth. Formerly the amount of carbon dioxid in the air was stated as 4 parts in 10,000, but repeated analyses with improved methods have shown that the correct amount is 3 parts or slightly more.[1] There is apt to be more carbon dioxid in the air just above the soil than at a height of 8 or 10 feet. This is not because the carbon dioxid is heavy and settles, but because the soil air usually contains more of this gas. Air collected at great heights by balloons has just the same percentage of CO_2 as air at sea level. The air over the sea contains somewhat less than air over the land. Carbon dioxid in the air comes

[1] Average of many analyses by F. G. Benedict is 0.031. *Carnegie Publications No. 166*, 1912.

from the oxidation of organic matter, from respiration, from fermentation, from chemical action in the soil, and from mineral springs. The exhaled breath contains about 4.4 per cent. of CO_2.

Even a small alteration in the percentage of carbon dioxid, either up or down, would throw out of adjustment a long-established balance, and this would alter the climate of the earth and might cause the death of all living beings. The carbon dioxid in the air is the source from which green plants with the assistance of sunlight obtain their carbon, and is thus indirectly the source of the carbon in the bodies of animals. The normal variations in the carbon dioxid of air in the open are too small to be of sanitary importance, and it is only when stagnant or inclosed air is polluted by combustion and respiration that we find accumulations which may have a bearing upon health. In narrow courts and in smoky air the free atmosphere may contain 0.7 to 0.8 per cent. Workshops may contain from 32 to 53 parts of carbon dioxid per 10,000, and breweries as much as 10 per cent. Its significance varies with its source. Enormous volumes of carbon dioxid are constantly being poured into the atmosphere. Manchester adds 8,000,000 cubic meters of CO_2 a day from the chimneys of industrial establishments. Even then the air of the city averages only 0.385 per cent. CO_2, while the air of the country averages 0.318 per cent.—a very slight difference. It is estimated that from all sources 500,000,000 tons are discharged annually into the atmosphere. The reason that the carbon dioxid does not accumulate and increase is that it is constantly removed, especially by growing vegetation. Plants absorb enormous amounts under the influence of light and chlorophyl to build carbohydrates. It has been estimated that an acre of tree land withdraws in one season about 4½ tons of CO_2. Much of the gas is also absorbed by water, which at ordinary temperatures takes up its own volume.

The amount of carbon dioxid produced by respiration varies with the vitality, size, and activity of the individual. During violent exercise almost ten times as much carbon dioxid may be discharged as during sleep. On the average a man discharges about 0.6 of a cubic foot of carbon dioxid per hour and a woman about 0.4 of a cubic foot. During ordinary activity a man produces, in round numbers, one cubic foot per hour. An ordinary gas jet burns about 6 cubic feet of gas per hour and produces about 3 cubic feet of carbon dioxid. Therefore, so far as CO_2 is concerned, a man vitiates the air less than a gas jet.

CO_2 as an Index of Vitiation.—For years the amount of CO_2 in the air has been generally adopted as the most convenient index of the total conditions which are usually prejudicial to health and comfort. The efficiency of ventilation also for years was usually determined by an estimation of CO_2.

CO_2 in itself is not irritating or poisonous. Large volumes may

be taken in beverages or inhaled without noticeable effects. Effects are first felt on the human system when the CO_2 reaches 2 or 3 per cent. Respirations increase with the percentage, both in frequency and depth, until about 5 per cent., when there is distinct panting, and at 7 or 8 per cent. the dyspnea becomes distressing; at 10 or 11 per cent. headache, nausea, and chilliness may be noted. Observations made by Professor W. G. Anderson in my laboratory show that these symptoms are more acute when the carbon dioxid is added to the air rapidly. Tolerance or second wind may be obtained in atmospheres containing even as much as 10 per cent. Animals soon die when the percentage reaches 35 or 45 per cent. in an artificial atmosphere. Man soon becomes unconscious and suffocates in an atmosphere containing 30 per cent. of CO_2.

Pettenkoffer in 1858 proposed 10 volumes of CO_2 in 10,000 volumes of air as the limit for inhabited rooms. De Chaumont (1875) found that an unpleasant odor becomes perceptible in air containing 6 volumes of CO_2 in 10,000, and fixed this as the limit, which for many years has been accepted by sanitarians. It was soon learned, however, that the percentage of CO_2 may rise much higher before ill effects become perceptible. Carnelley, Anderson, and Haldane in 1887 concluded that for the very crowded elementary schools a lower limit than 13 volumes was not practical. Haldane and Osborn in 1902 recommended a limit of 12 volumes for factories and workshops at the breathing level, and that when gas or oil is used for lighting the proportion should not exceed 20 volumes. The general consensus of opinion to-day is that 10 volumes in 10,000 is well upon the safe side, although, so far as CO_2 itself is concerned, more might be permitted without fear. Carbon dioxid is by no means the most mischievous of the constituents of vitiated air. It is not merely a waste product. It is one of the important hormones of the body. It regulates the action of the heart, influences the tonus of blood vessels, and stimulates the respiratory center.

It is certainly erroneous and unscientific to rely upon determinations of CO_2 in the air of a room as the sole measure of its condition for respiration. Carbon dioxid never accumulates sufficiently in any ordinary room to become in itself serious; further, the amount of CO_2 in the air of a room gives no indication whatever of the moisture, the temperature, or the motion of the air of that room. While the amount of CO_2, then, gives us a rough index of the degree of vitiation of the air, it affords no information concerning its physical conditions, which are of special importance.

If CO_2 stratifies at all in a room, more of it will be found nearer the ceiling than the floor, despite the fact that it is a comparatively heavy gas. This is owing to the fact that the CO_2 rises with the warmed expired breath and collects in the stratum of hot air near the ceiling. Diffusion takes place but slowly.

The significance of carbon dioxid upon health is further discussed on page 643.

Methods for Determining Carbon Dioxid.—For the ordinary purposes of a sanitary analysis it is not necessary to make an accurate analysis of the carbon dioxid in air, such as the chemical analyist or the student of metabolism would make in scientific research. As the carbon dioxid in itself is not poisonous and is only an imperfect index of other impurities, and as its significance varies with its source, sufficient information may be gleaned for sanitary purposes from methods that give results relatively comparable.

The most accurate method of determining CO_2 in the air is that described by Petterson, and used in the Petterson-Palmquist, the Sonden or the Haldane apparatus. Both the Petterson-Palmquist and the Haldane methods are convenient, practical, and sufficiently accurate for all ordinary purposes. The method of Cohen and Appleyard is reasonably accurate and very convenient. The methods of Wolpert and Fitz give only rough estimates.

Collection of Samples.—The collection of the samples of air to be analyzed is fully as important as the actual test. The following methods may be used:

The Water Siphon Method.—Two bottles (diameter one-third the height), volume about one-half liter, of nearly equal capacity, should be fitted with rubber stoppers carrying small glass tubing connected by several feet of rubber tubing with clamps. Fill one bottle completely with water, nearly free from carbon dioxid.

The pair of bottles is taken to the place from which the air is to be collected. The inlet or collecting tube may be long, so as to reach nearly to the ceiling, or short; if long, the first siphoning should be rejected to insure filling the inlet tube with the air desired. The stoppers are then exchanged and the sample taken. The air-filled bottle should be stoppered and taken to the laboratory; or the test solution may at once be added, and the bottle stoppered and shaken, noting minutes and seconds in the Cohen-Appleyard method. One bottle of water with a small reserve will serve for a number of takings before absorbing a sufficient amount of CO_2 to materially influence the results. If the water is acidulated it will take up less CO_2.

The steam vacuum method may be used as an alternative in less accurate work. The bottles should be of about 150 c. c. capacity, made for a ground-glass stopper, but fitted with a rubber stopper. These are filled with steam from water first freed of CO_2 and air by boiling for 5 minutes. The bottles are inverted and a steam jet having sufficient pressure to throw the vaporized steam at least one foot is allowed to fill the bottle for 3 minutes. Meanwhile a thin coating of vaselin is applied half way up the sides of the stopper. This not only

makes a tight joint, but facilitates removing the stopper. As soon as the collecting bottle is removed from the steam jet the stopper is instantly inserted and securely pushed in while the bottle is still in the inverted position. To test the method for completeness of vacuum hold the bottle in an inverted position under water at 70° F. and remove the stopper.

Samplers consisting of special glass tubes provided with a glass stopcock at both ends may be used to collect samples of air, particularly for the Sonden, Petterson-Palmquist, or Haldane apparatus. These samplers have a capacity of about 100 c. c.; some of them hold about 200 c. c. They must be clean and absolutely dry. The samplers are filled by means of a bulb from a Davidson syringe. Care must be taken that enough of the air to be examined is drawn through the sampler to force out all of the original air it contains. Samples may be collected in duplicate, and duplicate analyses are always advisable.

Standard Lime Water for Testing CO_2 (Used in the Cohen-Appleyard and also in the Fitz and Wolpert methods).—To a liter of distilled water add 2.5 c. c. of phenolphthalein (made by dissolving 0.7 gram of phenolphthalein in 50 c. c. of alcohol, and adding an equal volume of water). Stand the bottle of water on a piece of white paper and add, drop by drop, saturated lime water till a faint color persists for a full minute. Now add 6.3 c. c. of saturated lime water and quickly cork the bottle, or connect the pipette.

The Haldane Apparatus.—This apparatus, shown in Fig. 77, was introduced for the determination of carbon dioxid in the case of ordinary rooms, schools, factories, etc. As the apparatus is portable, the analysis can be made directly on the spot and the carrying to and fro of samples is thus avoided, if desired. If the burette is allowed to fill while the apparatus is carried across the room, a good average sample is obtained. As it takes some seconds for the mercury to run down, this method of taking the sample can easily be adopted, or a sampler containing the air to be examined can be connected directly by means of rubber tubing to the

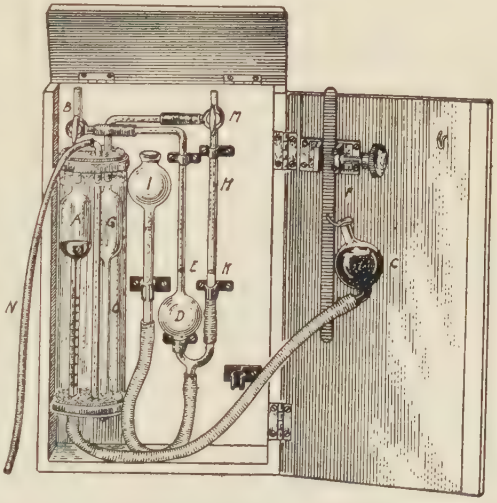


Fig. 77.—Portable Haldane Apparatus for Small Percentages of Carbon Dioxid.

gas burette. In this case it is advisable to discard the first filling of the gas burette A in order to get rid of the air in the rubber tubing and connections. About 4 minutes are required for an analysis. The accuracy is about 1 part in 10,000.

The air burette A, which is enclosed in a water jacket O, consists of a wide, ungraduated and a very narrow graduated portion. This is divided into 100 divisions, each of which corresponds to 1 part in 10,000. The lowest division is marked 0 and the numbering is upward from this point. Any difference between a reading at or near zero and a second reading is thus shown by the scale in volumes per 10,000, there being no calculations or corrections.

The absorption pipette D is filled to the mark E with a 20 per cent. solution of caustic potash through reservoir I. The control tube G enclosed in the water jacket is used to correct for variations in the temperature of the sample during the analysis. It is connected with the potash pipette D by the tube H, which has a mark K. The pressures under which the readings are made are maintained constant by adjusting the levels of the potash solution to the marks E and K. To compensate for variations of temperature of the water jacket O, air is blown through the tube N, thus agitating the contained water.

The technique of an analysis is summarized as follows:

(1). Open the 3-way cock B to the air to be examined and raise the mercury bulb C to expel the air in the burette A. Lower the mercury bulb and hang on the adjustable rack F so that the sample is drawn in and the level of the mercury falls to near the zero mark.

(2) Open the cock M to the air for a moment and then turn it so as to connect the control tube with the potash solution in the tube H.

(3) Turn the cock B so as to connect the sample with the potash pipette D.

(4) Squeeze the rubber tube of the potash reservoir I so as to raise the potash level about an inch above the marks E and K, and see that the level of the potash alters sharply and about equally in the two tubes.

(5) Blow air through the water jacket O.

(6) Raise or lower the potash reservoir I till the potash is exactly at the mark K in the tube H.

(7) Raise or lower the mercury bulb C by means of the arrangement F till the potash is exactly at the mark E.

(8) Read off the mercury level on the scale of the burette to 0.2 of a division. (First reading.)

(9) Raise the mercury bulb, so as to drive the air into the potash pipette D; then lower it a little and raise it twice again so as to wash any carbonic acid in the connecting tubing into the pipette.

(10) Return the air to the burette A.

(11) Again blow air through the water jacket.

(12) Squeeze the rubber tubing and adjust the two potash levels at K and E, as before, and again read off the mercury level. The first reading subtracted from the second gives the amount of CO_2 in volumes per 10,000.

(13) After the analysis open G to the outside air through cock M and shut off A from D by turning cock B. This will prevent fouling of the apparatus by the sucking up of the potash solution.

The Petterson-Palmquist Method.—This is a simplified Sonden apparatus by which the volume of CO_2 in the air may be determined directly in hundredths of a per cent. by volume. The method is accurate to one part in 20,000 of air, provided care is taken with the tests.

The principle is essentially the same as that found in the Haldane or the Sonden apparatus. A measured amount of air is collected in a gas burette. This volume of air is then transferred to an Orsat tube containing a strong solution (20 per cent.) of potash, which absorbs the CO_2. The air is then returned to the gas burette and remeasured for loss in volume. Great care must, of course, be exercised that the pressure and temperature are precisely the same before and after absorption. The gas burette A, Fig. 78, is first filled with mercury by raising the reservoir E. The sample to be analyzed is then drawn into A by lowering E. There must always be a drop of water on the surface of the mercury and also in the compensating cylinder C. In this way the air sample is kept saturated with moisture. In reading the volumes the meniscus of the mercury is each time so adjusted that the pressure in A is exactly the same as the pressure of the air in the compensating cylinder C. This is accomplished through a differential manometer containing a drop of colored liquid (petroleum, in which azobenzol is dissolved). This manometer is connected by capillary glass tubes on one side with A and on the other side with C. After the gas pipette A is filled with the sample of air to be tested, close the stopcocks D, F, C, and G and adjust the level of the mercury in A, so that the drop of liquid in the manometer stands at zero on the scale. This adjustment is accomplished through the set screw E. In this way the air in A may always be brought to the same pressure as that prevailing in the compensator C. Since the air in both compensator and pipette is, from the beginning of the experiment, separated from the external atmosphere by closing the stopcocks f, g, and c, variations in the external atmosphere have no effect. The temperature is regulated by filling the jar with water and keeping it agitated, preferably with bubbles of compressed air.

Each analysis consists of three operations:

(1) The air is drawn in from the outside and is measured, the level of the mercury in the graduated tube being brought to the zero

mark. The upper and narrower part of the scale, where each division denotes 1/10,000 of the volume of the pipette, is used in analyses of

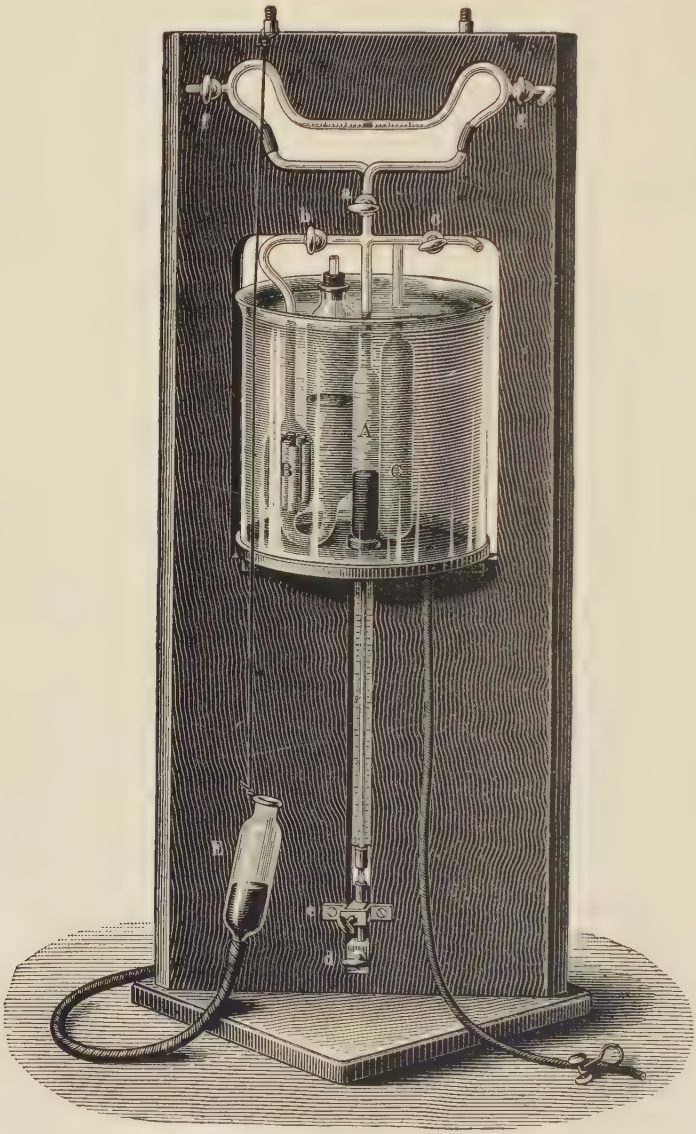


FIG. 78.—PETTERSON-PALMQUIST APPARATUS.

atmospheric air, or the ordinary air of rooms, where the per cent. of carbon dioxid is at the most not higher than 0.4 per cent. In the analysis of very impure air the lower part of the graduated tube is used, each

division here corresponding to 1/1,000 of the whole volume. In measuring the volume the stopcocks f, g, b, c, and d must be closed.

(2) The stopcocks d and b are opened, a is closed, and the air is passed from A to B. After one or two minutes the carbon dioxid is absorbed and the air may be brought back into A; b is then closed and a is opened.

(3) The mercury level in A is so adjusted that the index again takes its normal position. The decrease in volume is then read off on the scale.

Acidulated water may be used to expel the air from samplers into the burette of the gas analysis apparatus, if the operation is quickly done. If a refinement of accuracy is desired mercury is preferable, for even acidulated water will take up some CO_2.

Method of Cohen and Appleyard.—This method is based upon the fact that, if a dilute solution of lime water slightly colored with phenolphthalein is brought in contact with a sample of air containing more than enough CO_2 to combine with all the lime present, the solution will gradually be decolorized. The time necessary to discharge the color depends upon the amount of CO_2 present. The amount of lime water and the volume of air being constant, the rate of decoloration varies inversely with the amount of CO_2.

Collect samples of air in clean, clear glass-stoppered bottles of half liter capacity. The sample may be collected by exhausting the air from a bottle with a pair of bellows or by completely filling the bottle with water and then emptying it at the point where the sample is to be taken. Run in quickly 10 c. c. of the standard lime water. Replace the stopper; note time. Shake the bottle vigorously until the pink color disappears; again note time, and ascertain the corresponding amount of CO_2 from the following table:

Time in Minutes to Decolorize the Solution	CO_2 per 10,000	Time in Minutes to Decolorize the Solution	CO_2 per 10,000
1¼	16.0	3½	7.0
1⅓	13.8	4	5.3
1½	12.8	4¼	5.1
2	12.0	5	4.6
2¼	11.5	5¼	4.4
2¾	8.6	6¼	4.2
3¼	7.7	7½	3.5

Methods of Wolpert and Fitz.—These are rough methods for determining carbon dioxid, and, while not accurate, are useful because of their simplicity and convenience.

The volume of air that must be brought into contact with a definite

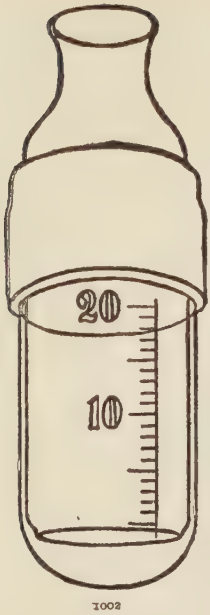


FIG. 79.—FITZ AIR TESTER.

quantity of lime water in order to neutralize all the lime is taken as a measure of the CO_2 in the air. The quantity of lime water and the time of reaction remaining constant, the amount of CO_2 varies inversely as the volume of air. The apparatus consists of graduated shakers, either Wolpert or Fitz (see illustration), and a pipette for measuring 10 c. c. of lime water.

In using these testers be sure the plunger slides easily, then remove it and place 10 c. c. of the lime water solution into the tube. Introduce the plunger and press it to the top of the solution, then withdraw it to the higher graduation. Close the mouth of the small tube in the Fitz apparatus, or the stem of the plunger in the Wolpert, with the finger, and shake vigorously for 30 seconds. The volume of air brought in contact with the lime water is 50 c. c. in the Fitz apparatus and 40 c. c. in the Wolpert. Remove the finger closing the small end, press the inner tube or plunger again to the top of the lime water in the Wolpert apparatus or to the point marked T in the Fitz apparatus, and draw it up again, thus admitting 20 c. c. of the air to be examined in the Fitz and 40 c. c. in the Wolpert. Again shake for 30 sec-

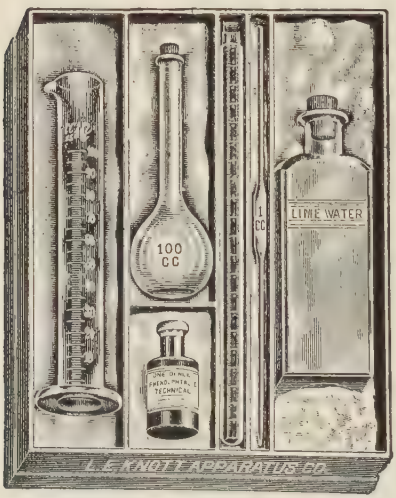


FIG. 81.—DEWING CO_2 APPARATUS.

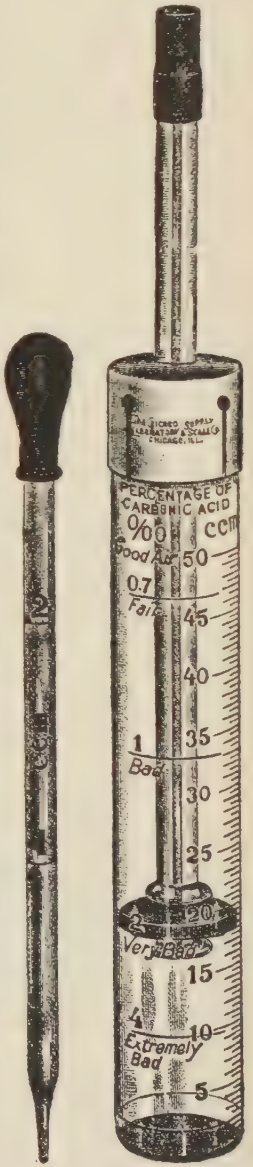


FIG. 80.—WOLPERT'S AIR TESTER.

onds. Repeat until the color is discharged. The first test will probably give an approximate result, and subsequent tests will serve to give more accurate data. From the volume of air used the amount of CO_2 can be determined from the table:

Air in c.c. Used	CO_2 per 10,000	Air in c.c. Used	CO_2 per 10,000
30	28	91	9
36	22	103	8
46	18	117	7
58	14	138	6
69	12	165	5
82	10	207	4

CHAPTER II

PRESSURE, TEMPERATURE, AND HUMIDITY

PRESSURE

Normal Atmospheric Pressure.—The pressure of the atmosphere at sea level is 15 pounds to the square inch, or, as indicated in the barometer, it will maintain a column of mercury 30 inches or 760 millimeters. A man of average size living at sea level is exposed to a total pressure of about 34,000 pounds—more than 15 tons. This great pressure must evidently have physiological importance. All the tissues and fluids of the body are subjected to this pressure and are in equilibrium with it. The interchange of gases on which life depends is largely a phenomenon of atmospheric pressure. The pressure of the air also keeps the heads of the bones in their sockets without muscular action, and doubtless performs other functions less obvious. The small variations in pressure such as occur day by day at sea level have no evident physiological effects.

Diminished Atmospheric Pressure.—A diminution in atmospheric pressure is equivalent to breathing rarefied or diluted air. The most important physiological effects of diminished atmospheric pressure are due to a diminution in the amount of oxygen absorbed, hence the breathing is deeper and the pulse rate quickened. As the altitude increases there is a lowered tension of oxygen in the alveolar air and a diminished tension of carbon dioxid. While the rate of respiration may be variously influenced in different circumstances, the depth of respiration is almost invariably increased. This of itself not only facilitates the oxygen supply, but also increases the elimination of carbon dioxid. Formerly a great compensatory increase in the number of red blood cells was believed to take place as a result of prolonged residence in high altitudes. Thus, assuming the average number of red blood cells per cubic millimeter at sea level to be about 5,000,000, at Davos (elevation 1,560 meters) the number of red blood cells averages 5.500,000 to 6,500,000. At Cordilleras (altitude 4,392 meters) the average number of red corpuscles is 8,000,000. A similar change in the blood has been produced by keeping rabbits and guinea pigs in rarefied air at sea level. According to Bürker, only a comparatively small increase takes place, amounting to 4 or 5 per cent., at altitudes of five or six thousand feet. The same moderate results have likewise been noted lately for much higher

altitudes. The higher figures of earlier workers are now accounted for by the more rapid evaporation of blood samples at higher altitudes, so that with improved technic the belief in the great increase in the oxygen-carrying blood constituents disappears.

At a height of 18,000 feet the pressure of the atmosphere is only half the pressure at sea level, thus:

Altitude.	Height of Barometer.
0 foot	30 inches
910 feet	29 "
1,850 "	28 "
2,820 "	27 "
3,820 "	26 "
4,850 "	25 "
5,910 "	24 "
7,010 "	23 "
8,150 "	22 "
9,330 "	21 "
10,550 "	20 "
13,170 "	18 "
16,000 "	16 "
18,000 "	15 "

"The highest dwelling place continuously occupied is the Observatory El Mirti, in the Andes, at 5,880 m. The Observatory at Arequipa is at 6,100 m. Thok djalung is a village in the Himalayas at 4,980 m. In Peru, Bolivia, and Northern Chili a very large part of the population live above 3,000 m. Potosi, which has numbered 100,000 inhabitants, is at 4,165 m., Cerro de Pasco at 4,350 m., the mines of Villacota at 5,042 m., the railway from Callao to Oroya culminates in a tunnel at 4,760 m., almost the height of Mont Blanc. An annual fair is held at Gartok, at 4,598 m., in the Himalayas, to which thousands annually come."[1]

It is evident that man may become adapted to breathing a rarefied air at great heights, which would overcome persons if the change were made suddenly from sea level.

The symptoms produced by a marked diminution in atmospheric pressure vary with circumstances. The effects are increased by cold, active muscular exertion, or improper clothing. The noticeable symptoms are increased rapidity of respiration and acceleration of the circulation, noises in the head and dizziness, impairment of the senses of sight, hearing, and touch, dulness of the intellectual faculties, and a strong desire to sleep. Sudden changes to a rarefied atmosphere cause syncope, weakness, dyspnea, dizziness, and nausea. These threatening symptoms sometimes go by the name of mountain sickness. Bert and Journet believe this condition is due to lack of oxygen and the symp-

[1] Leonard Hill: "Recent Advances in Physiology."

toms may, in fact, be relieved by adding oxygen to the air inspired. Bert kept a bird alive in oxygenated air, even though the pressure was reduced to less than 0.1 of an atmosphere. Kronecker concludes that mountain sickness is caused by a congestion of the lungs, impeding the flow of blood through them. Mosso and his followers attribute the physical disturbances of a reduced atmospheric pressure to the fact that the blood loses carbon dioxid more quickly than it loses oxygen, and attributes mountain sickness to this decrease of carbon dioxid in the blood (acapnia). Cohnheim believes there is a concentration of the blood at high altitudes; in fact, insignificant increases have been found by competent observers. The climate in high altitudes is always dry and evaporation proceeds rapidly. As a result individuals lose water more readily than at lower levels. If this explanation is tenable, an increase in corpuscles and hemoglobin content are in no wise the expression of lack of oxygen, but are rather the outcome of the increased evaporation under the altered conditions of climate.

The limit at which life may be sustained is about 26,000 feet, at which height consciousness is lost. At this height the barometric pressure of the air is 251 mm., which represents a pressure of oxygen of 52, which is the equivalent of 6.8 per cent. oxygen. P. Bert remained 20 minutes in a pneumatic chamber with a pressure of only 248 mm. without serious inconvenience.

Increased Atmospheric Pressure.—While man is often exposed to rarefied air, he is seldom subjected to increased pressure except under artificial conditions, such as in diving bells, diving suits, and caissons. The increase in atmospheric pressure in the deepest mines has little physiological significance. Divers and workers in caissons are not subjected to more than about 4½ atmospheres, and work under such pressure for only a few hours at a time. When a diving bell is lowered 10 meters into the water the air contained in it is compressed to one-half its original bulk, and the pressure of the air is accordingly doubled. Each 10 meters' depth means an additional pressure of one atmosphere. At a depth of 30 meters, about 100 feet, a diver is exposed to a pressure of 4 atmospheres or about 60 pounds per square inch. Bert exposed dogs to a pressure of 10 atmospheres, and then slowly released them without harm.

The physiological effects of an increased atmospheric pressure are mainly due to an increase in the amount of atmospheric gases (especially nitrogen) which are taken up by the blood, and also an increase in the chemical absorption of oxygen by the red blood cells. The serious consequences usually result from too rapid decompression.

Caisson Disease.—The effects produced by compressed air in caissons are: (1) those caused when the men are undergoing pressure, and (2) during or after decompression.

The symptoms produced by an increase of atmospheric pressure are a slowing of the respiration, which is evidently compensatory, but on account of compression of intestinal gases the respirations are deeper; the pulse is slower, and evaporation of water-vapor hindered. The voice may be altered; pains in the ear are common, due to pressure upon the drum, and may be obviated by swallowing air and thus passing it up the Eustachian tube into the middle ear. Sometimes the ear drum ruptures; headache and dizziness may also occur. During compression the blood keeps absorbing the gases of the air until the tension of the gases in the blood becomes equal to that in the compressed air. As soon as this equilibrium has been attained relief from immediate troubles is secured.

It is during and after decompression that the greatest danger to health and even risk of life occur. The most frequent symptom is excruciating pains in the muscles and joints, called by the workmen "bends." These pains may continue for a few hours or for two or three days. Occasionally there is bleeding at the nose; also severe abdominal pain, and vomiting, nausea, vertigo, dyspnea, and unconsciousness. Death may result from internal hemorrhage, or paralysis may ensue—the so-called diver's palsy.

The effects of increased atmospheric pressure and too rapid decompression were carefully studied by Paul Bert in 1878, who showed that the lesions are caused by the escape of gases of the atmosphere which have been taken up in excessive amounts, and are released in the blood and tissues when the pressure is diminished. The blood vessels may contain air emboli, which may lodge in vital parts and cause sudden death, or the delicate capillaries may break, leading to hemorrhage with resulting paralysis. Air emboli may be distressing or dangerous if they occur in the labyrinth of the ear, in the spinal cord, in the brain, or in the heart or other vital parts.

The prevention of caisson disease consists in gradual decompression. Sometimes the symptoms come on several hours after the workman has left the caisson. As soon as symptoms come on the workman should at once be hurried back into the compression chamber until equilibrium between the internal and external pressures is restored. He may then be allowed to pass through the decompression chambers, but very gradually. A medical air-lock should be provided at the works, well heated, and furnished with bunks and emergency supplies.

Barometers.—The pressure of the air is measured by means of barometers, the principles of construction and use of which are so well known that they do not require special description.

MOVEMENTS OF THE ATMOSPHERE

Moving air is necessary for the maintenance of health and is a prime requisite of good ventilation. The motion of the air serves the twofold purpose of bringing us a fresh supply and taking away the sewage-polluted air from our immediate vicinity. Moving air also favors evaporation and helps to prevent heat stagnation by keeping the surface temperature within normal limits. Paul, Heymann, and Erclentz, in Flügge's laboratory, and also Leonard Hill in England, emphasized the importance of moving air in assisting the heat regulation of our body. They believe that this is a much more important function of moving air than simply the bringing of fresh air or the carrying away of the products of respiration. In still air the body soon becomes surrounded by a warm, moist aerial envelope which causes an overheating of the surface of the body and results in the familiar symptoms of "crowd poisoning." In a still atmosphere we are soon surrounded by a blanket of stagnant and impure air, whether indoors or outdoors.

Much of the benefit of mountain, seaside, and other health resorts is attributable to the breezes that blow almost continuously at such places. The health of large cities located upon the seacoast or the shores of great lakes is favored by the quantities of moving air with which they are frequently flushed. A healthful climate is always a breezy climate —within reasonable limits. Much of the benefit of driving, of fanning, and of rocking-chairs is due to the motion of the air thus engendered.

If the air in a poorly ventilated room can be kept in motion, say with an electric fain, many of the ill effects of a vitiated atmosphere are avoided, for the products of respiration are diluted, and evaporation and heat interchange are favored. Thus, Leonard Hill placed eight students in a small sealed chamber which held about three cubic meters. He states that "at the end of half an hour they had ceased laughing and joking and their faces were congested. The carbon dioxid had gone up to 4 or 5 per cent. Three electric fans were then turned on, which merely whirled the air about just as it was. The effect was like magic; the students at once felt perfectly comfortable, but immediately the fans were stopped they again felt as bad as before." The relation of moving air to temperature and moisture, with reference to ventilation, is further discussed on page 647.

In nature the atmosphere is kept in almost constant motion as a result of differences in temperature. Thus, the hotter air in the tropics rises and divides into two currents, which flow toward the north and south, while heavier, colder air rushes along a lower level from the north and south to take the place of the lighter currents. The cold

currents from the poles are known as the trade winds, and the upper, warmer currents to the poles as the antitrades. The upper currents to the poles run northwest and southwest; while the lower currents from the poles run northeast and southeast.

The chief cause of periodic winds, such as daily sea breezes and monsoons, is the difference in the heating of the air above land and above sea. On a small scale the same principle is seen at play in theaters, churches, cathedrals, and public buildings. The great mass of people crowded together heats the air about them and it ascends; cool air rushes in from the aisles to take its place, hence the almost unavoidable drafts in such places.

The velocity of air currents is customarily measured by means of recording anemometers. These instruments require a considerable velocity of air and should never be used without a carefully prepared table of corrections whereby their readings may be adjusted.

It often becomes desirable in sanitary investigations, particularly in studies of ventilation, to determine the strength and direction of currents of air which are too delicate to be measured by means of anemometers. Lighted candles will show the direction of slight air currents, the flame being deflected in the direction in which the current is moving. More delicate than this is the method of noting the course taken by smoke from a joss-stick, cigarette, or cigar.[1]

When a current of air at the temperature of 55° to 60° F. moves at a rate of one mile per hour, there is no perceptible draft. The rate of movement in relation to our perception is as follows:

Air moving at 1.5 feet per second—1.0 mile an hour—imperceptible.
Air moving at 2.5 feet per second—1.7 miles an hour—barely perceptible.
Air moving at 3.0 feet per second—2.0 miles an hour—perceptible.
Air moving at 3.5 feet per second—2.3 miles an hour—draft.

The movement of warm air is less perceptible than the movement of cool air.[2]

TEMPERATURE OF THE AIR

The temperature of the air depends mainly upon solar and terrestrial radiation. The air absorbs vast quantities of heat from the sun, and as the heat of the earth is radiated into space a certain amount is again absorbed by the atmosphere. Accordingly, the air both keeps the heat out and keeps it in. It makes the days cooler and the nights warmer. "It is a parasol at noon and a blanket at night." Except for it there would be much more violent changes in temperature (Macfie).

The power of the air to absorb heat and to store heat depends largely on its humidity; that is, on the amount of water vapor it con-

[1] For a further discussion of this subject see "Air Currents and the Laws of Ventilation," by W. N. Shaw.

[2] For Drafts see page 175.

tains, for water vapor is opaque to heat rays. The water vapor is also a great reservoir of latent heat. When water evaporates a tremendous amount of latent heat is carried up into the atmosphere with it and again becomes actual heat when the vapor condenses. The quantity of heat thus stored up in water vapor is almost incredibly great.

Air expands $\frac{1}{491}$ of its volume for each degree rise of temperature; air at 32° F. and 30 inches barometric pressure is usually taken for unit of volume. A cubic foot of dry air at 32° F. and 30 inches barometer weighs 566.86 grains; at any other temperature, therefore, its weight can be ascertained by dividing by its increased volume.

The temperature of the air has a very important bearing upon health. Man has an almost incredible power of adapting himself to wide variations of temperature. Workers in foundries have sometimes to endure a heat of 250° F. and even higher for short periods of time. Temperatures of —75° F. are met with in polar expeditions. This is a range of at least 325° F. The reason that man, as well as other animals, is able to maintain a constant body temperature when exposed to such great variations of atmospheric temperature is due not only to the physiological mechanism which regulates heat production and elimination, but to the layers of air immediately in contact with the skin. We wear clothes to protect ourselves from external heat or cold, but still more do we wear air for that purpose. That is why very high temperatures are better borne when the air is in motion, which facilitates evaporation, than when the air is still, while extremes of cold are better borne when the air is still, for then we become clothed in a warm blanket of air. The effect of heat upon health, however, cannot be considered alone, for it depends on the humidity as well as on the movement of the air. Extremes of heat and cold are much more trying when the air is humid than when the air is dry.

It is of first importance that the arrangements for heating rooms, offices, schools, etc., should be so regulated that the temperature never exceeds 21° C. (70° F.); especially should this control be exercised in public rooms, such as schools, etc. As a rule, the temperature of heated rooms should be 17° to 19° C. (62.6° to 68.2° F.). The effect of temperature upon health is so closely interwoven with humidity that this relationship is discussed on page 613.

Methods of Recording Temperature.—*Mercurial or bimetallic thermometers* are best suited to take the temperature of the air. The most accurate mercurial thermometers for this purpose have an elongated bulb of mercury at one end and a ring at the other, through which a cord can be tied; the scale should be etched upon the glass. A good thermometer of this type generally is accurate to about one-half to one-fifth of a degree. Thermometers placed upon a backing of metal, card, or wood, with the scale painted upon the backing, are more orna-

mental than accurate. They usually possess a decided lag and are, therefore, not trustworthy. Thermometers should be suspended freely in the atmosphere or at least placed in a current of air sufficient to insure good ventilation about the mercury column.

Registering thermometers are of two principal types: those which record maximum and minimum temperatures, and those which make a continuous record of the changes of temperature that occur.

The maximum and minimum temperatures furnish but limited information, and, as such self-recording thermometers are almost invariably mounted upon a backing, they consequently have a considerable lag. They are only dependable where fluctuations in temperature are not rapid. Under these circumstances they may be used to record the highest and lowest temperatures.

For an intelligent understanding of the sanitary condition of any room or inclosed space neither single determinations nor maximum and minimum records are sufficient. Recording thermometers should be placed at various selected points and records should be obtained covering a period of several days. The best type of recording thermometers depend upon the movements of bimetallic bars, so arranged that as they contract and expand they cause a penpoint to bear upon a moving paper scale, and so leave an ink trace. The clockwork is generally wound up for a week, for which period the paper scale is also adapted.

HUMIDITY

Aqueous Vapor.—Water in its gaseous state is always present in the atmosphere. Water vapor is the most variable of the normal constituents of air, and also one of the most important, on account of its influence upon health. It is usual to consider water vapor apart from the other gases of the atmosphere, although it is just as much a gas as oxygen or nitrogen, and conforms to the general laws that govern the behavior of gases. As water vapor weighs only about three-fifths the weight of air, dry air is heavier than moist air under equal conditions of temperature, pressure, etc. It is customary to speak of air "holding" water vapor. As a matter of fact, the air has nothing to do with it, for it should always be clearly observed that the presence of water vapor in any given space is independent of the presence or absence of air in the same space. The amount of aqueous vapor which a space contains depends entirely upon the temperature and not upon the presence or the pressure[1] of the air. At 32° F., for instance, the air can "hold" 1/160 of its weight of water vapor, at 59° F. 1/80 of its weight, at 86° F. 1/40 of its weight. Roughly, every 27° F. increase of temperature doubles the amount of water vapor the air can hold in propor-

[1] A high barometer retards evaporation, while a low atmospheric pressure accelerates it. All volatile liquids evaporate instantly in a vacuum.

tion to its weight. In this way the heat of the atmosphere is self-protective, for it loads the air with water vapor, which in turn absorbs much of the heat. The latent heat is again given off on condensation. The actual amount of water vapor which the air can hold at different temperatures is shown in the following table:

A cubic foot of air can hold at		
10° F.	1.1	grains
20° "	1.5	"
30° "	2.1	"
40° "	3.0	"
50° "	4.2	"
60° "	5.8	"
70° "	7.9	"
80° "	10.0	"
90° "	14.3	"
100° "	19.1	"

As the temperature rises in arithmetical progression the power to retain vapor increases with the rapidity of a geometric series having a ratio of two.

The amount of water vapor in the air may be expressed either by: (1) its *vapor tension.* The tension of the water vapor in the air is expressed in inches or millimeters of mercury. If a drop of water is placed in a vacuum, say in a barometer tube, some of the water vaporizes and the mercury is depressed, owing to the tension of the water vapor. The amount that evaporates, as well as the tension, depends upon the temperature. (2) Its weight per unit volume of air, i. e., the *absolute humidity;* and (3) the ratio of the amount of water vapor in the atmosphere to the amount it could hold at the temperature in question if saturated; that is, the *relative humidity.* Complete saturation of the air with moisture is stated at 100, and lesser amounts by percentages. (4) The amount of water vapor in the air may also be found from its *dew-point.* The dew-point for any temperature and humidity is the temperature to which the air may be cooled when precipitation takes place.

The vapor tension or the absolute humidity indicates how much water vapor the air contains, while the relative humidity is an expression of how much vapor it might contain. The amount of water vapor which air can hold when saturated at different temperatures has been calculated and recorded in Glaisher's hygrometric tables.[1] It is, therefore, very easy, by referring to these or to the tables in the U. S. Weather Bureau—*Bulletin No. 235*—to calculate the relative humidity if we know the actual humidity or the dew-point or *vice versa.*

[1] The standard hygrometrical tables in use the world over are those prepared by Mr. James Glaisher, F. R. S., of the Royal Observatory, Greenwich, England.

The amount of moisture which out-of-door air ordinarily contains varies from about 30 per cent. or less to saturation.

In meteorological tables, giving climatic particulars of any town or

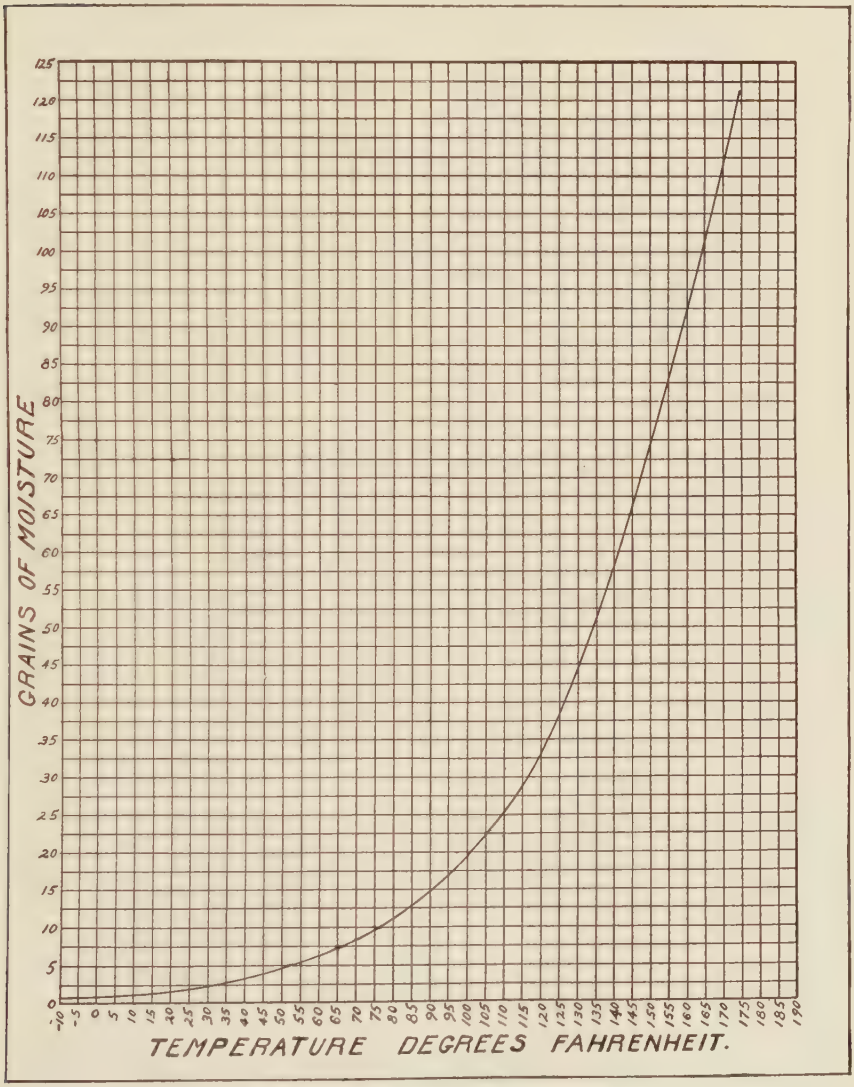


Fig. 82.—Diagram Showing Absolute Humidity in Grains at Different Temperatures.

locality, the relative humidity is usually stated; but it should be noticed that the relative humidity bears no constant relationship to the absolute humidity. As the relative humidity varies greatly throughout the day, and as the readings are not always taken at the same time of

day in different localities, it at once becomes evident that comparisons are not reliable. In fact, a moist or dry climate cannot be predicted from the relative humidity. Thus, the mean relative humidity of Davos is as high as 79 per cent., whereas it is generally known that the climate of Davos is dry. On the other hand, in Egypt the average relative humidity is very low, although this country is known to have a moist climate. This is for the reason that the humidity readings in Egypt are taken from 10 A. M. to 6 P. M., and vary from 30.5 per cent. at Assouan to 51.7 per cent. at Menahouse. As a matter of fact, the relative humidity in Egypt decreases from 100 per cent. at dawn to 22 per cent. at noon, and may be quickly altered to the extent of 50 per cent. by a warm wind. The humidity, therefore, through the hot, sunny daytime is not a measure of the climate, so far as moisture and dryness are concerned.

In England the relative humidity averages 75 per cent. In California it drops from 100 per cent. at dawn to 22 per cent. at noon. A hot wind, by increasing the capacity of the air for moisture, may also lower the relative humidity very quickly. Thus, the Föhn wind when it reaches the Riviera lowers the relative humidity 50 to 60 per cent. in an hour or two. The mean relative humidity of Denver for the year is only 42 per cent., at San Diego, on the coast, 72.9, at Los Angeles, a few miles inland, 66.6. In the heart of the Libyan desert the relative humidity may be as low as 9 per cent. At the seaside daily variations in humidity are less than inland (Macfie). The air in forests is 10 or 20 per cent. more humid than air in the open. There may be a very great difference in the relative humidity of outside cool air and of air in a closed heated room, in that the latter may be very much dryer.

So far as the effect of humidity upon health is concerned Dr. Huggard well states: "The really essential point is not the amount of moisture, relative or absolute, that is present, but the amount that can still be taken up. This varies enormously with the same degree of relative humidity at different temperatures, as the following table from Renk will show:"

AMOUNT OF VAPOR THAT CAN STILL BE TAKEN UP AT DIFFERENT TEMPERATURES AND THE SAME RELATIVE HUMIDITY.

Temperature	Relative Humidity	Absolute Humidity Grams per Cubic Meter	Grams of Vapor that can still be taken up
	Per Cent.		
−20° C.	60	0.638	0.426
−10° C.	60	1.380	0.920
0° C.	60	2.924	1.950
10° C.	60	5.623	3.749
20° C.	60	10.298	6.866
30° C.	60	18.083	12.056

We see by this table that the same expression, 60 per cent. relative humidity, might be applied to air capable of taking up 0.426 gram or 12.056 grams of vapor, and thus the expression as a measure of the drying capacity of the air is obviously misleading.

Dr. Huggard gives a second very instructive table, the obverse of the above:

Temperature	Relative Humidity	Vapor: Grams per Cubic Meter	
		Present	Capable of Still Being Taken up
	Per Cent.		
3° C.	0	0	6
10° C.	36	3.4	6
15° C.	53	6.8	6
20° C.	65	11.2	6
25° C.	73	16.9	6
30° C.	80	24.1	6

We see from this second table that air with relative humidities of 0, 36, 53, 65, 73, and 80 per cent., and containing quantities of water vapor varying between 0 and 24.1 grams per cubic meter, are all capable of further taking up exactly the same amount of vapor. Again the expression of relative humidity is misleading.

When the relative humidity reaches 80 to 85 per cent., moisture condenses and begins to show upon objects in rooms. This influences natural ventilation through porous building materials.

There may be a very marked difference between the humidity of indoor and outdoor air, owing in part to the condensation of moisture, especially in winter, upon the cold walls and windows.

The differences between external and internal humidities depends largely upon the temperature of the surfaces in the room. These surfaces, though apparently dry, may, in fact, hold moisture in large quantities; the walls and ceilings may contain more water than all the air in the room. Ordinarily there is a continual exchange of moisture between the air and the room surfaces. In this way the walls serve as a compensating reservoir to help maintain the humidity of the air approximately constant. Cold walls, cold windows, and cold surfaces generally condense the moisture from the air so rapidly that great difficulty is experienced in raising the relative humidity of the air of a room under these circumstances.

The humidity in the air is influenced by altitude. The higher we go the air becomes colder and rarer and, therefore, able to contain less moisture. Its absolute humidity, therefore, decreases. Half of the total water vapor of the atmosphere is below 2,000 meters. On the

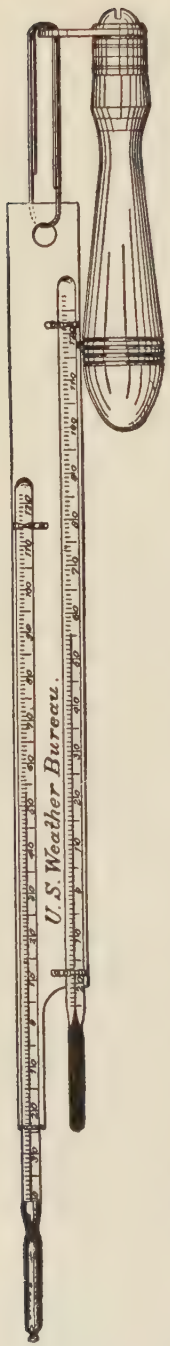


Fig. 83.—Sling Psychrometer.

other hand, the relative humidity shows no regular change with change of altitude. Clouds do not necessarily imply high relative or absolute humidity of the lower atmosphere. Rainfall also gives only a very general indication of the humidity of the atmosphere. A place with high rainfall may have low absolute and relative humidity, and *vice versa;* that is, a rainy district is not necessarily a damp district, so far as the atmosphere is concerned. Dew also bears no constant relationship to the humidity of the atmosphere, for a clear sky and a dry atmosphere favor its formation. Air containing mist is obviously moist.

Methods of Determining Humidity in the Air.—The amount of water vapor in the air may be determined either by (1) weighing, (2) psychrometers or hygrometers, (3) the dew-point.

Weighing.—The amount of moisture in the air may be determined by passing a given volume of air through a tube or flask containing an hygroscopic substance, such as calcium chlorid or sulphuric acid. If sulphuric acid is used small flasks are filled with pieces of pumice which have been heated to a high temperature over a Bunsen burner, and dropped while hot in concentrated sulphuric acid, removed, and quickly drained.

The increase in weight represents the amount of moisture in the volume of air passed through the flasks, or the absolute humidity. Knowing the temperature of the air, it is then easy to determine the relative humidity by reference to tables of maximum water capacity for certain volumes of air at varying degrees of temperature.

Psychrometers.—The most convenient of all methods for measuring atmospheric moisture is to observe the temperature of evaporation, that is, the difference between the temperatures indicated by wet and dry bulb thermometers. The United States Weather Bureau regards the sling psychrometer as the most reliable instrument for this purpose. In special cases rotary fans or other means may be employed to move the air rapidly over stationary thermometer bulbs.

The *sling psychrometer* consists of a pair of thermometers provided with a handle, which permits them to be whirled rapidly (see Fig. 83). The bulb of the lower of the two thermometers is covered with thin muslin, which is wet at the time an observation is made.

RELATIVE HUMIDITY TABLE

Air Temperatures.	Difference Between the Dry and Wet Thermometers																														Air Temperatures.
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	
30	100	89	78	67	57	47	36	26	17	7																						30
35	100	91	82	73	65	54	45	37	28	19	12	3																				35
40	100	92	84	76	68	60	53	45	38	30	22	16	8	1																		40
45	100	92	85	78	71	64	58	51	44	38	32	25	19	13	7	1																45
50	100	93	87	80	74	67	61	55	50	44	38	33	27	22	16	11	6	1														50
55	100	94	88	82	76	70	65	59	54	49	43	39	34	29	24	19	16	10	6	1												55
60	100	94	89	84	78	73	68	63	58	53	48	44	39	34	30	26	22	18	14	10	6	2										60
65	100	95	90	85	80	75	70	65	61	56	52	48	44	39	35	31	28	24	20	17	13	10	6	3								65
70	100	95	90	86	81	77	72	68	64	60	55	52	48	44	40	36	33	29	26	23	19	16	13	10	7	4	1					70
75	100	95	91	87	82	78	74	70	66	62	58	55	51	47	44	40	37	34	31	27	24	21	19	16	13	10	7	5	2			75
80	100	96	92	87	83	79	75	72	68	64	61	57	54	51	47	44	41	38	35	32	29	26	23	20	18	15	13	10	8	6	3	80
85	100	96	92	88	84	80	77	73	70	66	63	60	56	53	50	47	44	41	38	36	33	30	28	25	22	20	17	15	13	11	9	85
90	100	96	92	88	85	81	78	75	71	68	65	62	59	56	53	50	47	44	41	39	36	34	32	29	26	24	22	20	17	15	13	90
95	100	96	93	89	86	82	79	76	72	69	66	63	60	58	55	52	49	47	44	42	39	37	35	32	30	28	25	23	21	19	17	95
100	100	97	93	90	86	83	80	77	74	71	68	65	62	59	57	54	51	49	47	44	42	39	37	35	33	31	29	27	25	23	21	100
105	100	97	93	90	87	84	81	78	75	72	69	66	64	61	58	56	53	51	49	46	44	42	40	38	35	33	31	30	28	26	24	105
110	100	97	94	90	87	84	81	78	76	73	70	67	65	62	60	57	55	53	50	48	46	44	42	40	38	36	34	32	30	28	27	110
115	100	97	94	91	88	85	82	79	76	74	71	69	66	64	61	59	57	54	52	50	48	46	44	42	40	38	36	34	33	31	29	115
120	100	97	94	91	88	85	83	80	77	75	72	70	67	65	62	60	58	56	54	51	49	47	45	44	42	40	38	36	35	33	31	120
125	100	97	94	91	88	86	83	80	78	75	73	70	68	66	64	62	59	57	55	53	51	49	47	45	43	42	40	38	37	35	33	125
130	100	97	94	91	89	86	83	81	78	76	74	71	69	67	65	62	60	58	56	54	52	50	49	47	45	43	42	40	38	37	35	130
135	100	97	94	92	89	86	84	81	79	77	74	72	70	68	65	63	61	59	57	55	53	51	50	48	46	45	43	41	40	38	37	135
140	100	97	95	92	89	87	84	82	79	77	75	73	71	68	66	64	62	60	58	56	55	53	51	49	48	46	44	43	41	40	38	140
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	

Fig. 84.

This muslin covering should be kept in good condition and should be frequently renewed. It is also desirable to use pure water. The so-called wet bulb is thoroughly saturated by dipping it into distilled water. The thermometers are then whirled rapidly for 15 or 20 seconds, stopped, and quickly read, the wet bulb first. This reading is kept in mind, the psychrometers immediately whirled again and a second reading taken. This is repeated three or four times or more, if necessary, until at least two successive readings of the wet bulb are found to agree very closely, thereby showing that it has reached its lowest temperature. A minute or more is generally required to secure a correct reading. The psychrometer should not be whirled in the direct rays of the sun, and if used out of doors the observer should face the wind. It is a good plan, while whirling the instrument, to step back and forth a few steps to further prevent the presence of the observer's body from giving rise to erroneous observations.

In correcting psychrometric observations the atmospheric pressure at the time must be obtained, and the results deduced from the tables based on a pressure nearest that observed. The difference in the temperature between the wet and the dry bulb is computed to the nearest tenth of a degree. Having the temperature and the pressure of the air and the depression of the wet bulb, it is only necessary to read directly from the tables the dew-point, the vapor pressure, and the relative humidity. These tables will be found in *Bulletin No. 235* of the United States Weather Bureau. A condensed table is given in Fig. 84.

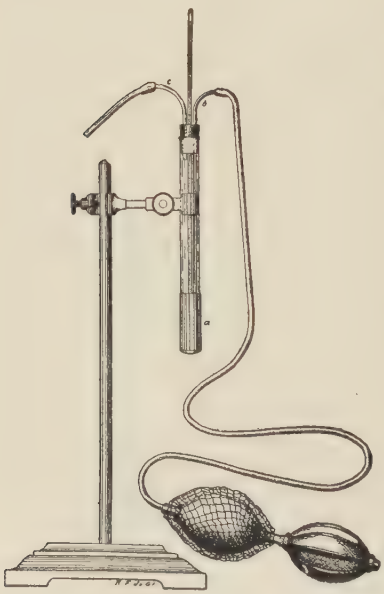


Fig. 85.—Dew-point Apparatus.

The Hair Hygrometer.—This apparatus depends upon the expansion and contraction of a suitably prepared hair under the influence of moisture. It can be made a reasonably accurate instrument, and some types are arranged for continuous record. One of the principal difficulties with hair hygrometers is that a sufficient current of air does not always come in contact with them.

The Dew-point.—The dew-point may be obtained by direct observation from Regnault's apparatus, shown in Fig. 85. This instrument consists essentially of a thin polished silver tube *a*, cemented upon the lower end of a longer glass tube, as shown. The stopper closing the upper end of

the glass tube is fitted with two lateral tubes of hard rubber *b* and *c*, and also carries a delicate thermometer, the bulb of which is placed near the center of the silver tube. The tube *b* extends to the bottom of the silver tube; *c* projects but a short distance through the cork. A rubber aspirating apparatus, as shown, is connected with the tube *b*, and a long tube joined to *c* serves to carry off the fumes. The apparatus is held in a clamp faced with cork or other non-conducting substance.

Observations are made by filling the silver cup with ether or similar volatile liquid, which is caused to evaporate and cool the silver cup by manipulating the aspirating bulb. At the proper point of cooling a deposit of dew is seen to form on the polished silver surface. The object is to ascertain accurately the temperature at which the dew will just deposit. It is necessary that the temperature be lowered very slowly at the critical point, also that there be plenty of liquid in the cup, and that it be agitated sufficiently to have a uniform temperature throughout, and, finally, the surface of the silver must be perfectly clean and in a favorable light, so that the faintest deposit of dew is at once visible. The temperature shown by the thermometer at this moment may be regarded as the temperature of the dew-point. Knowing the dew-point, the humidity of the air may be found by reference to the above-mentioned tables.

Relation of Humidity and Temperature to Health.—The physiological significance of moisture in the air varies with many factors, but especially with temperature. In a general way it may be said that moist air is depressing and enervating, while dry air is tonic and stimulating; also that cold air is tonic, while warm air is depressing. The human body can adapt itself to wide variations in heat and humidity, and by means of suitable clothing and food the range may be greatly increased. Various combinations of heat and humidity may be trying or even hurtful; the most mischievous combinations are cold damp air and warm moist air, also an excessively dry air, especially when artificially warmed. Many climates in which people are reasonably healthy have a relatively high humidity, and some regions famed for their salubrity are notoriously dry and arid. The frequently changing temperatures and variable amounts of water vapor of most climates may be beneficial in stimulating the heat-regulating mechanism.

The temperature and humidity of the air affect health mainly by influencing the heat-regulating mechanism of the body. More heat is produced within the body than is required, hence heat must be lost, else heat stagnation or heat stroke will result. The temperature of the air, but still more its humidity, influences heat loss. It will, therefore, be necessary to briefly review the mechanism by which the constant temperature of the body is maintained.

The chief source of the body heat comes from the food we eat. Approximately 80 per cent. of the food we eat is used to furnish heat to maintain the body temperature, while only about 20 per cent. furnishes energy in the form of motion. Heat is lost from the body chiefly in two ways: (1) by *heat transfer,* or loss by radiation, conduction, and convection; (2) by *evaporation,* chiefly by the evaporation of the water of perspiration. The loss by heat transfer diminishes as the temperature of the surrounding air rises. The temperature of the body would rise when the atmospheric temperature went above 70° F. were not perspiration then secreted. So long as the perspiration can evaporate freely the heat production and heat loss are balanced. With a high humidity evaporation is lessened and the balance is maintained by rushing blood to the skin, which causes an elevation of the temperature of the surface, and thus the loss of heat by radiation, conduction, and convection is facilitated.

Humidity influences the output of heat from the body in two ways: (1) it increases the conductivity of atmosphere for heat—a cooling influence—hence cold moist air is chilling; (2) it interferes with evaporation of perspiration—a heating influence—hence warm moist air is enervating. There is a neutral zone, around 68° F., at which humidity has comparatively little effect. Hence, if the temperature of a room is kept just right and the occupants are sitting still, it makes little difference whether the air is humid or dry. However, a difference of a few degrees above or below this temperature will have a marked influence.

Rubner and his coworkers showed that the evaporation of water from the body cannot be regarded as being dependent merely on the percentage humidity of the atmosphere. The temperature of the layer of air in contact with the body is the factor of great importance. Thin clothes and still air, under certain conditions of external temperature, may favor evaporation, while nakedness and moving air favor conduction and radiation. The heat-losing mechanisms of the body are adjustable to varying conditions within wide limitations, so that diminished loss by evaporation is compensated for by increased loss by conduction and radiation.

The amount of moisture in the air conducive to health and well-being is often stated to be somewhere between 50 and 75 per cent. relative humidity. These figures may be very misleading. There is no such thing as a normal humidity, for the amount of moisture in relation to health depends upon the temperature, clothing, motion of the air; also upon diet and muscular activity and other factors. Neither the relative humidity nor the absolute humidity nor the temperature of the air alone is a satisfactory guide as to its condition in relation to health. One factor gives the sanitarian scant information; however,

the temperature as registered upon the wet-bulb thermometer is most significant.

Importance of the Wet-bulb Temperature.—The individual susceptibility to temperatures depends entirely on the temperature recorded by the wet-bulb thermometer,[1] no matter what the dry bulb registers. Hill, Rubner, Pembrey, Boycott, Cadman, Nagel, and practically all authorities agree with Haldane that the air of workrooms should not exceed 70° F. by the wet-bulb thermometer.

Rubner states that an untrained man can be in comfort in a temperature of 75° F. and 80 per cent. humidity (wet bulb about 70° F.) only when he is quiet. At 73.4° F. and 60 per cent. humidity he found a resting man lost by evaporation 75 grams of water per hour, and at 84 per cent. humidity (wet bulb 70° F.) only 19 grams. These figures show that three-quarters of the heat loss may be maintained by conduction and radiation when the wet bulb reaches 70° F.

Cadman concludes that at:

72° wet bulb....Inconvenience is experienced, unless heavy clothing is removed and light clothing worn.

78° " " ....Little inconvenience is felt if considerable bare body surface is exposed. Hard work is much facilitated if a perceptible current is passing over the body.

82° " " ....If clothes be removed, and maximum body surface exposed, work can be done providing current of air is available.

85° " " ....Body temperature becomes affected, and only light work is possible.

Boycott made the following significant observations upon himself:

"At rest and stripped I found that my body temperature rose rapidly if the wet bulb exceeded 88° to 90° F. with a dry bulb of about 100°, though no rise occurred with a dry bulb of 110° and wet bulb of less than 85°. I have on many occasions spent periods of about an hour in doing ordinary laboratory work in air with the dry bulb at 95° and the wet bulb at about 65° without any material discomfort. If, however, the wet bulb rises to 88° to 90°, one's body temperature begins to go up, even when completely at rest, and one becomes exceedingly uncomfortable and on occasions feels very ill. These sensations can be, to some extent, remedied by local cooling of the skin (e. g., cold water on the head), but the rise of body temperature is progressive and must eventually end in heat-stroke."

A man is much less efficient in a warm moist atmosphere; hence it is an advantage to both employer and employee that work be performed at temperatures below 70° F. by the wet bulb. At the lower tempera-

[1] One of the thermometers of a psychrometer is known as the wet bulb. See page 610.

tures work is done faster, more efficiently, and with less fatigue, discomfort, and injury to health. To work in a warm moist atmosphere increases the temperature, pulse, and loss of moisture out of proportion to the work done. It is the master's pockets which suffer under such conditions, for the workers instinctively avoid the discomfort of overheating themselves through lessened exertion.

Effects of Warm Moist Air.—When air above 88° F. becomes saturated evaporation can no longer compensate for decrease in radiation, and the body temperature accordingly rises and heat-stroke may ensue. The injurious effects of the summer heat are practically always the result of combined heat and humidity.

According to Rubner and Lewaschew, when the air is very humid the heat loss by evaporation is very much lessened, and, accordingly, at 80 per cent. humidity and temperature of 24° C. (75.2° F.) becomes after a time insupportable to a man unaccustomed to it, and exposure to it is only possible with complete muscular rest. If, however, the air is very dry a temperature of 24° to 29° C. (75.2° to 84.2° F.) can be usually endured. These temperatures are often exceeded in the summer time in America. By practice a certain amount of accommodation to the effects of a hot moist climate may be acquired.

There is no known serious injury to health caused by working in a warm moist air, provided that a considerable rise of body temperature is avoided. The effects of heat and moisture may be diminished by light clothing, bare legs and arms, whereby the loss of heat from the skin is increased.

Working in moist, overheated rooms has the further disadvantage of wetting the clothes with perspiration, which causes discomfort, dirt, and untidiness, and liability to chilling the surface on going outdoors.

A poorly ventilated room in which the air becomes vitiated is usually a warm moist atmosphere, and the ill effects of a vitiated atmosphere are mainly caused by the heat and moisture.

Effects of Cold Damp Air.—When such air is injurious the victim is usually underclad, improperly fed, or has been living an indoor life. In certain cases cold damp must always be injurious, as, for instance, where the vital forces are at a low ebb and where there is restricted capacity for making heat, such as infancy or old age; in cases of kidney disease, where hindrance of evaporation means extra work for the kidneys; also in cases where there is a tendency to rheumatism or disorders of metabolism. The effects of cold damp air may be neutralized by proper clothing, by muscular activity, and, to a limited extent, by diet.

Just how cold damp air influences health is not well understood. It throws an added load upon the heat-producing mechanism to maintain the body temperature; the strain falls especially upon digestion

and metabolism, and also upon the circulation and the kidneys, and indirectly upon the nervous system. Macfie suggests that: "Dry air quickens metabolism both through its cooling and drying capacity, while damp air slows it by diminishing loss of water. It is possible that much of the harm attributed to damp and to cold is due to a depression of metabolism and accumulation of harmful waste products in the body." Dr. H. I. Bowditch in 1862 formulated the law of soil moisture, and believed that tuberculosis was more common over moist soils than dry ones. According to our present conception, the relation between dampness or moist soil and tuberculosis is quite indirect; if there is any connection it is due merely to the fact that the combination of cold and dampness depresses vitality and thereby lowers resistance.

A healthy man may daily move in and breathe cold damp air without suffering in health to any appreciable extent; however, it is generally believed that a cold damp air predisposes to affections of the respiratory passages, to rheumatism, and neuralgias.

Effects of Warm Dry Air.—A relatively dry air feels better than moist air at most temperatures. The stimulating and pleasant effects of a dry climate can only be appreciated by one who has visited an arid region—such as our southwestern plateau. However, when air is abnormally dry, especially if warm, the evaporation from the body is greatly increased. Thus, Rubner and Lewaschew found that a man weighing 58 kilograms gave off the following amounts of carbon dioxid and moisture in one hour at different temperatures in dry and moist air:

	Dry Air.			*Moist Air.*		
Temp.	Relative Humidity of Air.	CO_2	H_2O	Relative Humidity of Air.	CO_2	H_2O
15° C.	8%	32.2 gm.	36.3 gm.	89%	34.9 gm.	9.0 gm.
20° C.	5%	30.0 gm.	54.1 gm.	82%	28.3 gm.	15.3 gm.
25° C.	6%	31.7 gm.	75.4 gm.	81%	31.4 gm.	23.9 gm.
29° C.	6%	32.4 gm.	103.3 gm.			

Air that is warm and at the same time abnormally dry, such as that produced by furnace heat, causes an excessive loss of moisture and concentration of the fluids in the tissues and organs of the body. Man consists of 58.5 per cent. of water. A very small percentage of loss may be serious; when the percentage reaches 21 per cent. death results. The warmed and dried atmosphere of our overheated houses gives a sense of chilliness, owing to excessive evaporation, and favors irritation and infection of the respiratory mucous membranes. If a room at 68° F.

is not warm enough for a healthy person, we may be sure that it is because the humidity is too low.

The problem of constructing buildings in such a way as to keep the interior up to a fair degree of humidity is a large one. So far engineers have made little practical progress toward its solution. Satisfactory devices may be had to improve the moisture in large public buildings, but these devices have so far proved too expensive for private dwellings, offices, or schoolrooms.

The humidity in living rooms may be improved by setting about growing plants and porous dishes, such as flower pots full of water. If such receptacles are set near electric fans evaporation is facilitated. Pans or pots of water may also be placed upon the radiator.

CHAPTER III

MISCELLANEOUS

Odors.—Odors in a living room come mostly from human sources. The sources of these odors are: foul breath, decaying teeth, unclean mouths, nasal catarrh, sudoriferous glands, especially those of the pubes, feet, and axillæ, also gases from the stomach and bowels. The decomposition of matter on the skin and also in the clothes adds a very disagreeable odor, accentuated in a warm moist atmosphere. The peculiar odor in some rooms, especially sick rooms, seems to be none of these: just what constitutes the somewhat characteristic man-smell is not known.

While odors may be very unpleasant, they are not known to seriously influence health; contrary to common opinion, they are not by any means a reliable sign of danger. The presence of bacteria or dust in the atmosphere has no special relation to odors. Some poisonous gases, such as carbon monoxid, are practically inodorous.

The air of inhabited rooms ordinarily must be quite full of various scents which we do not appreciate, either because our sense of smell is not keen enough, or because we have become so accustomed to them that they are not noticed. An atmosphere that does not appear to be unpleasant while remaining in a room may seem intolerable upon returning to it after a period in the fresh outdoor air. Man's sense of smell is not keen when compared to that of some of the lower animals; nevertheless it is extremely sensitive to certain odors. Thus, it can determine 0.000,000,03 gram of musk. The acuteness of the sense of smell varies markedly in different individuals.

When a room smells stuffy and close it may be taken as a fairly reliable index that the air is vitiated; this is especially true in a clean room not complicated with odors from clothing and sources other than man. In fact, the odors observed upon entering a room from the outside fresh air often furnish better evidence of imperfect ventilation than laboratory tests.

De Chaumont made accurate observations and found that when the CO_2 amounts to 6 parts per 10,000 the atmosphere begins to smell

close and stuffy. Pettenkoffer found air containing 7.5 parts of CO_2 per 10,000 from the expired breath to have a marked odor, and 10 parts a very unpleasant odor. With a little practice various grades of vitiated air can be detected up to 10 or 12 parts of CO_2 per 10,000.

The odors from marshes and from decomposing organic matter are not apparently hurtful. One of the most famous stenches that has been recorded, if not the most famous, was that which arose in 1858 and 1859 from the Thames, which at that time was grossly polluted with the sewage of London (Sedgwick). Dr. Budd insisted that no very serious results followed. After giving his proof Budd[1] states: "Before these inexorable figures the illusions of half a century vanished in a moment." We now know that odors in the air bear no reference to contagion or infection and, however unpleasant, need not be feared as such. Sewer "gas" is discussed on page 638.

The effect of odors upon health is not at all understood. When we sense a pleasant smell we involuntarily take deeper breaths; on the other hand, unpleasant odors diminish the respiratory exchange. The latter are accordingly harmful to that extent and the former stimulating. Odors influence the nervous system in various ways; some stimulate, others depress psychic activity; some odors have a well-known influence upon sexuality. Occasionally odors are so disagreeable that they induce nausea, even vomiting. It is remarkable how quickly we may become accustomed to odors, but because our sense of smell has been dulled is no guarantee that the cause of the odors may not continue to produce its effects. Leonard Hill thinks that it is very doubtful if the unpleasant smelling exhalations of the bodies of men have any ill effects on men accustomed to them, and not of esthetic temperament.

Light.—All the rays of the sun pass through the atmosphere before they reach the earth.[2] The air acts as a differential filter, holding back many rays, especially those of shorter wave-length; that is, the ultraviolet end of the spectrum. These rays have marked chemical power. Bunsen and Roscoe investigated this question of the atmospheric absorption of the chemical rays of the sun, and came to the conclusion that in passing through the atmosphere the ultraviolet rays lost about 66 per cent. of their chemical power. We have already seen that many of the heat rays are also absorbed by the atmosphere. "More heat and we might be roasted, more light and we might be

[1] Dr. William Budd: "Typhoid Fever: Its Nature, Mode of Spreading, and Prevention," pp. 148-151. London, 1873. This is a remarkable contribution which the student is advised to read.

[2] The waves of light are not waves of the atmosphere, but of the ether; however, they are absorbed, reflected or refracted by the dust and moisture contained in the air. It is convenient to consider light, as well as electricity and radioactivity, at this point.

blinded, more chemical energy and we might be slain like the microbes.

The rays of shorter wave-lengths have chemical and photodynamic powers which must have an important relation to health. These rays act upon photographic negatives; hasten the hatching of flies' eggs and frogs' eggs; they sunburn the skin; they kill many bacteria, including the tubercle bacilli; they cause heliotropism; they combine chlorin and hydrogen into hydrochloric acid; they cause the oxidation of oxalic acid and other chemical reactions; they blacken silver salts. It has been shown that in buckwheat poisoning (fagotoxismus) these actinic rays play an important part. The skin eruptions upon the exposed surfaces in pellagra are also explained upon the photodynamic theory, that is, the poison is believed to be activated by certain light rays.

The air as a filter of the sun's rays bears a very important but little understood relation to life. It is now well known that some of the sun's rays have intense chemical and "vital" power. We know something about the chemical rays, the luminous rays, and the calorific rays, but there are doubtless many ether vibrations of which we know nothing. Macfie speculates that, "even, indeed, as the crops of the northern zone outstrip the crops in the south of France, so at certain times may the activity of nations be stimulated or depressed by atmospheric variations affecting the composition of solar radiation."

The physiological action of light is just beginning to receive the serious attention it deserves. We are all familiar with the calming effect of the dim religious light of churches and the stimulating effect of the glare of the theater. The intense light of the tropics and of high altitudes is believed in some way to bring on nervous disorders, but the relation is but vaguely understood. Some of the ill effects of rooms, attributed to bad air and poor ventilation, are due in part to the over-stimulation of excessive illumination.

Method for Measuring Illumination.—The method which is recommended as a standard procedure depends on the use of photosensitive paper, such as can be obtained from any dealer in photographic materials. By exposing the sensitized paper through a slot in a cardboard for a sufficient period of time, and noting the number of seconds or minutes consumed to match in depth a standard shade of color, the intensity of light can be determined with accuracy. If a fresh piece of paper is exposed to the direct rays of the sun for three seconds it will assume a shade which can be used as a standard for a given series of tests. The intensity of light at other points may be compared with this by noting the number of seconds required to color a fresh piece of paper from the same lot to the same shade.

Electricity.—The question of electricity is also a question of vibra-

tions, not of the air, but of ether, and one shrouded in much obscurity. The electric potential of the air varies considerably. It is highest in winter and lowest in summer, and shows diurnal variations. It is increased by winds and is especially increased by the condensation of vapor. It also increases as we ascend.

It is assumed that electric changes in the air and in other objects surrounding us exercise an influence on health and vitality, but the influence is obscure and mainly a matter of conjecture.

Radioactivity.—Soon after the discovery of radium by the Curies it was proved, chiefly through the investigations of Elster and Geitel, that the air and soil and certain mineral springs contained radioactive substances. Newly fallen rain and snow are also radioactive. Air drawn from the soil by means of a pipe, or air shut up in underground cellars and caverns, is specially radioactive, as is also the air on mountain tops. The air in clear weather has greater radioactivity than in dull weather.

Certainly radioactive substances have important physiological, physical, and chemical effects. They ionize the air, rendering it a conductor of electricity; they cause a fluorescence of certain chemical substances; they produce a sensation of light if they strike the eye; and if too active may cause destruction of living tissue. Substances so potent must have some physiological influence.

Smoke.—Smoke is a product of combustion and consists of a mixture of gases containing solid particles. Ordinary smoke consists largely of unburned carbon particles, hydrocarbons, and other pyroligneous products, gases, some of them poisonous, such as carbon monoxid, also mineral acids, etc. Angus Smith gives the following analysis of smoke from a common house fire:

Smoke from a Common House Fire

	Carbon Dioxid	Carbon Monoxid	Oxygen	Nitrogen
Gas from chimney 4 feet above the fireplace	0.35	.....	16.93	80.02
	1.65	0.38	19.29	78.68
Gas from the middle of a good fire.	19.46	0.09	.....	80.45
A great mass of coal over the fire,	20.90	0.10	.....	79.00
the gas taken from below the glowing mass	17.50	.....	0.60	80.04
	17.44	.....	0.39	82.17
A heap of glowing coal, gas taken close to spot where carbonic oxid was burning	15.43	3.49	0.96	80.12
	18.17	2.48	.....	79.35
Gas from clear fire below	16.10	.....	4.95	78.95
Gas from the same fire at upper part, 1 inch below the surface	17.21	.....	4.25	78.54
	18.20	0.99	.....	78.21

Dr. Cohen of the Manchester Air Analysis Committee gives the following analysis of soot collected from the roofs of glass houses in Kew and Chelsea:

	Chelsea Per Cent.	Kew Per Cent.
Carbon	39.0	42.5
Hydrocarbons	12.3	4.8
Organic bases (pyridins, etc.)	2.0	....
Sulphuric acid	4.3	4.0
Hydrochloric acid	1.4	0.8
Ammonia	1.4	1.1
Metallic iron and magnetic oxid of iron	2.6	....
Mineral matter (chiefly silica and ferric oxid)	31.2	41.5
Water not determined (say difference)	5.8	5.3

Large manufacturing chimneys are the chief offenders. There are two main causes of smoky chimneys: (1) insufficient boiler capacity, and (2) improper stoking. The cure of the smoke nuisance consists in the installation of boilers of sufficient power so that they need not be forced, and the use of mechanical stokers. The electrification of railroads and the more general use of electric power generated from water pressure help materially to lessen the amount of smoke in cities.

The London County Council permits black smoke for five minutes after the lighting of furnaces. Other towns allow as much as 15 minutes. Most laws distinguish between black smoke and white smoke, although the one is about as pernicious as the other.

In Boston the density of the smoke is graded into four classes, in accordance with Ringelmann's chart. This is a rather complicated system, depending upon the character of the stack, the density of the smoke, and the time, as shown in Fig. 86.

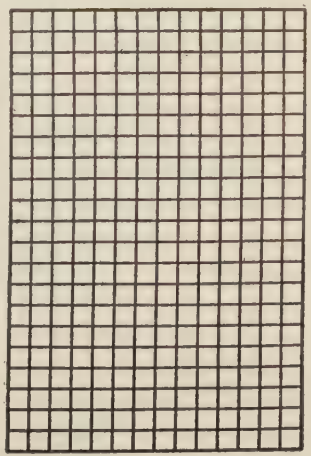
The amount of smoke in some manufacturing centers is almost incredible. Dr. W. N. Shaw estimates that London gives to the atmosphere every day about 7,000,000 tons of smoky air containing over 400 tons of soot, and he calculates that smoke deprives London of about one-sixth its possible sunlight and daylight in summer and about one-half its possible sunlight and daylight in winter.

The injurious effect of smoke on health has perhaps been overestimated. It acts directly and indirectly. Directly it irritates the mucous membranes of the upper respiratory passages, and Asher and also Rubner believe that it increases the mortality from acute pulmonary diseases. They state that smoke and soot predispose to acute pulmonary tuberculosis. Indirectly smoke is a source of dirt and general nuisance and leads to depression of the spirits. It shuts out the light, soils with soot, and deters the opening of windows in order to let in fresh air. The presence of mineral acids in the air has a corrosive in-

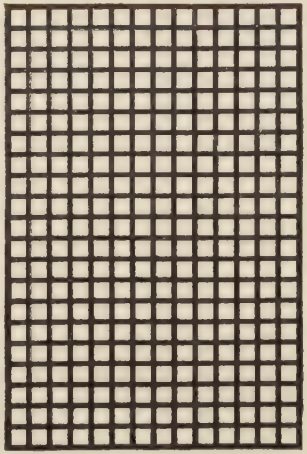
FIG. 86.—TABLE SHOWING THE DENSITY OF SMOKE, IN ACCORDANCE WITH THE RINGELMANN CHART, WHICH MAY BE EMITTED FROM THE VARIOUS CLASSES OF STACKS IN BOSTON, MASS., AND THE DURATION OF SUCH EMISSION

CLASSES	1		2		3		4		5		6		LOCOMOTIVES MOVING TRAINS OF SIX CARS OR MORE	
	Chart No.	Mins.	Chart No.	Mins.	Chart No.	Mins.	Chart No.	Mins.	Chart No.	Mins.	Chart No.	No. seconds in 5-minute periods	Chart No.	No. seconds in 5-minute periods
1910	3	6	4	5	4	10	4	9	4	12	3	40	3	50
1911	3	4	3	10	3 including 4	20 including 5	3	12	3	15	3	30	3	40
1912	2	8	3	6	2 including 3	30 including 10	3	7	3	9	3	20	3	30
1913	2	6	3	3	2 including 3	25 including 5	3	3	3	5	3	20	3	30

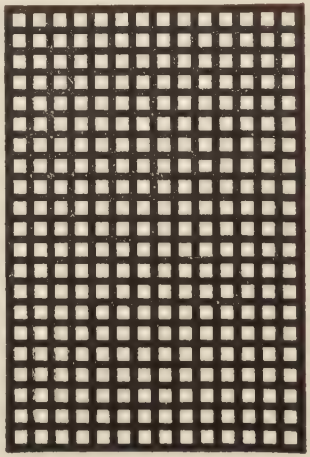
Reduced Copy of Ringelmann Chart



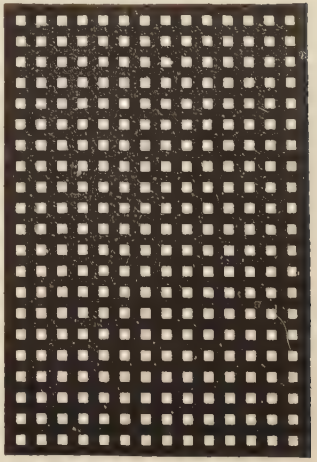
1. Equivalent to 20 per cent black



2. Equivalent to 40 per cent black



3. Equivalent to 60 per cent black



4. Equivalent to 80 per cent black

INSTRUCTIONS FOR USING THE RINGELMANN CHART.—Hang smoke chart on a level with the eye, about 50 feet from observer, as nearly as possible in line with chimney. Glance from smoke to chart and note corresponding number, recording same and time of observation. Repeat observations at one-fourth or one-half minute intervals. From these records the average density may be determined for each hour or for each day. No smok is reecorded as No. 0. 100 per cent black smoke is recorded as No. 5. Experienced observers often record in half chart numbers.

fluence upon inorganic substances, and doubtless acts injuriously upon plant and animal life. The economic losses from the soiling action of soot are enormous. Even if it were not injurious to health, smoke is so evident a nuisance that communities are justified in every effort to check and prevent this growing abomination.

Smoke polluted with poisonous chemical vapors may be quite serious. Thus, hydrogen sulphid, found in large quantities in the smoke generated in sulphate of ammonia and tar works and from alkali wastes, is a poisonous gas. The arsenical vapors given off chiefly from lead and copper smelters kill vegetation for wide areas around.

Fog.—Fogs are caused by the condensation of water vapor on particles of dust. Dust particles have a varying capacity for condensing and attracting moisture, depending upon their power of radiating heat and on their affinity for water. Carbon dust is hygroscopic and, therefore, encourages fogs. The ammonia and sulphuric acid in smoky air also occasion and aggravate fog. The air of manufacturing cities, therefore, possesses all the elements to form a fine persistent fog which forms a "chemical pall" between the city and the sky.

The more carbon a fog contains the blacker it is. The general result of a fog is to shut out sunlight and fresh air and to "partially suffocate unfortunate citizens in clouds of noxious chemicals." Fog contains all the irritating properties of smoke in a concentrated form, and it also in a measure prevents the escape of the city-made carbon dioxid. The CO_2 in the city air during a fog may rise to 10 parts per 10,000. If smoke is bad fog is ten times worse. It has been shown that during city fogs sickness increases and the death rate rises. From the economic standpoint fog causes greater financial losses than smoke. Russell calculates the annual loss to the people of London from fog to total about $9,000,000 a year. The main items in this loss consist in extra washing, including extra soap, the damage to dresses, curtains, carpets, and textile fabrics, the replacing of wall-papers, and the painting of houses, the restoring of gilt and metal work, the slow destruction of granite, marble, and stonework of buildings, the extra cost of artificial illumination, etc. This estimate does not include the losses resulting from its action on health.

Dust.—Dust is not only a nuisance, but under certain conditions is known to be prejudicial to health. Dust is in reality a normal and very important constituent of the air; it exists everywhere in the atmosphere and profoundly affects some of the physical conditions of our environment. One of the most important functions of dust is to limit the humidity of the air by causing the precipitation of moisture in the form of rain, and to help control temperature by the formation of clouds, mists, and fogs. Aitken, who has made a special study of this subject, says that without dust "every blade of grass and every branch of tree

would drip with moisture deposited by the passing air; our dresses would become wet and dripping, and umbrellas useless; but our miseries would not end here. The insides of our houses would become wet; the walls and every object in the room would run with moisture." Without dust there would be no rain, no clouds, no mist, for the water vapor which condenses upon each particle of dust forms the nucleus of a raindrop.

Dust disperses the light and decreases the transparency of the atmosphere, especially if the atmosphere be also humid. What is known as haze is really dust carrying a minute amount of moisture.

Although dust particles are universally present in the known atmosphere, they are very irregularly distributed. Organic dust exists only in the lower strata, while inorganic particles are found wherever the air has been examined. Ordinarily there is more dust indoors than in outdoor air. The size of the dust particles varies enormously, from gross masses to microscopic and ultramicroscopic particles. The vast numbers and universal presence of these particles may be realized by examining a sunbeam. Air free of dust is an artificial product obtained only with special care and in small amounts in the laboratory.

Most of the dust is torn from the earth by the winds; much of it comes from the carbon and other particles in smoke; considerable amounts consist of minute grains of salt derived from sea spray; and great quantities are added by volcanoes. Finally, the air contains interplanetary particles which fall through it in a constant shower.

The spectrum shows the bands of sodium everywhere in the atmosphere. This is lifted into the air by the wind from the sea spray. The water evaporates, leaving the salt particles to float about at the will of the wind.

Organic dust consists of the dry and disintegrated particles which are blown into the air from the animal and plant kingdoms. They consist of epithelial scales, seed, spores, bacteria, pollen, plant cells, fluff of various kinds, bits of insects, starch, pus cells, algæ, rotifers, fragments of hair, feathers, and bits of tissue, fibers of cotton, etc.

The inorganic dust, which is derived mostly from the soil, from the sea, and from interplanetary space, consists chiefly of silica, aluminium silicate, calcium carbonate, calcium phosphate, magnesia, iron oxid, sodium chlorid, etc.

Dust particles may be carried enormous distances by the winds. Ehrenberg detected organisms belonging to Africa in the air of Berlin; and fragments of infusoria belonging to the plains of America in the air of Portugal. The smoke of the burning of Chicago reached to the Pacific coast. The volcanic dust of Krakatoa, consisting chiefly of glassy pumice, was found for years in our atmosphere, and it is assumed that some of it may have traveled several times around the

world. Macfie has seen in the Canary Islands clouds of dust sufficient to obscure the sun, though the dust had come all the way from the African mainland. All of us living on the Atlantic seaboard have seen the yellow days caused by forest fires several thousands of miles away.

Dust and Disease.—"Normal" atmospheric dust, free from bacteria, causes no appreciable irritation of the healthy respiratory mucous membranes. Dust becomes injurious when excessive in amount or when irritating in character, or when it contains injurious microorganisms; the injury also depends upon the constancy of its presence and somewhat upon the susceptibility of the individual.

Dust may act indirectly as a predisposing cause of many infections, as well as directly irritating and inflaming the respiratory passages. The statement that dust opens the door to tuberculosis and other infections of the air passages, such as common colds, influenza, pneumonia, etc., can no longer be questioned. We must first limit ourselves to a consideration of the effect of dust free of noxious bacteria; in the next section we will discuss the question of bacteria in the air.

The general effect of mineral dust breathed for a long period of time is to cause an irritation of the mucous membranes and an inflammatory condition of the lung tissue. The term *pneumonokoniosis* is a general name for affections of this kind. The term is modified according to the various kinds of dust. Thus, *anthracosis* is caused by coal dust; *siderosis* by iron or steel dust; *silicosis* or *chalicosis* by stone dust; *byssinosis* by cotton particles or vegetable fiber dust.

In certain cases the dust is retained as deposits in the lungs and neighboring lymph glands without further damage. The lungs and bronchial glands of all adults are more or less discolored from particles, which are constantly inhaled. The particles are taken up by the phagocytes and deposited in the lymphatic spaces of the lung or carried to the neighboring lymph glands, where they are enmeshed. Under certain circumstances the dust irritates the delicate structures and leads to infections and destruction of tissue. Thus, we hear of stone mason's phthisis, steel grinder's phthisis, and potter's rot. Among the dusty trades may be mentioned pottery and earthenware manufacture, cutlery and file-making, certain departments of glass-making, copper, iron, lead, and steel manufacturing, stone-cutting, chimney-sweeping, textile trades, etc. Oliver ("Diseases of Occupation") examined the atmosphere in which the brushers-off, the finishers, and the porcelain-makers generally work, and found it to contain 640 million particles of dust per cubic meter of air, while several of the finishers, i. e., the persons whose work consists in removing the excess of the dried glaze on the ware, are often breathing an atmosphere containing 680

million particles of dust to the cubic meter. It is little wonder that bronchitis and phthisis are common.[1]

Dust consisting of inorganic particles is more harmful than dust consisting of organic particles, because the former are sharper and more irritating. House dust is more harmful than outside dust, not only because there is more of it, especially in badly ventilated and ill-kept rooms, but because it is more apt to contain living pathogenic bacteria. House dust may be kept down by cleanliness and avoidance of dry dusting and sweeping; the use of vacuum cleaning; and by a free system of ventilation. Much house dust is blown in from the outside, and some of it comes in on dirty shoes. In buildings ventilated with a mechanical system the air may be filtered through bags or passed through a water curtain, which will eliminate much dust. Oiling floors with a wax or paraffin mixture helps to keep down indoor dust. Carpets tacked down are sanitary abominations and should be replaced with rugs that permit outdoor cleaning and sunning.

Street dust contains coal dust, metallic dust from the operation of trolley cars, material swept from houses and from shaking rugs from windows, the grinding up of roadbeds by vehicles, ashes, and other materials blown from barrels and teams; the bacteria are derived from dried fecal matter from horses and other animals, dried sputum, the soil, and a variety of other sources. Street dust may contain pathogenic organisms, such as the tubercle bacillus, many varieties of cocci, the colon bacillus, *Bacillus aerogenes capsulatus,* and possibly, under special conditions, tetanus, malignant edema, and occasionally other pathogenic microorganisms. Street dust, therefore, becomes more than a nuisance, for it is not only irritating, but may be a source of infection.

To keep down street dust requires, first of all, a well-constructed road with a good surface, oiled or properly cared for; the control of animals; the covering of ash barrels and carts hauling dusty loads; the use of automobile vacuum cleaners to replace the old or the present-day methods of dry sweeping. Attention must also be given to spitting on sidewalks and streets, the enforcement of smoke ordinances, the more extensive flushing of streets, and general attention to cleanliness.

The pollen of certain plants flying in the air as dust leads to hay fever in susceptible individuals (see Anaphylaxis, page 407).

Methods for Examining Dust.—Petri Dish Method.—The simplest and one of the most useful methods of determining the amount of dust and its composition is by means of suitable receptacles, such as Petri dishes, upon which the dust is allowed to settle for a sufficient period of time to enable a considerable quantity to accumulate.

[1] For a discussion of the dusty trades, see chapter on Industrial Hygiene.

Particles are then examined under the microscope, or, if desired, they can be gathered upon a watch glass and weighed.

Weighing.—The air may be passed through cotton or filters of other material, the quantity of air being measured either by means of a gas meter or other device and the increase in weight of the filter determined. Whatever the filtering medium the quantity of air should be large, in order that the quantity of dust may be appreciable in amount and be fairly representative. By weighing the filtering material before and after passing the air through it, the aggregate weight of dust in the quantity of air taken for examination can be determined. It is necessary to guard against increase in weight of the filtering material through the absorption of water. This can be done by placing the filtering material in a desiccator before and after filtration and just before weighing in each case.

The Koniscope.—The koniscope, invented by Professor John Aitken, consists of two brass tubes connected at right angles and suitably fitted with stopcocks and a small air pump. By exhausting the air from one of the tubes, allowing the space to become saturated with water vapor by evaporation from wet blotting paper within, and then allowing this moisture to condense upon the dusty atmosphere under examination, clouds of different degrees of density will form inside the tube. The approximate density of the clouds can be measured by looking through the tubes, windows being provided for this purpose. A table is supplied with the instrument to give the approximate number of dust particles corresponding to clouds of different degrees of density.

CHAPTER IV

BACTERIA AND POISONOUS GASES IN THE AIR

BACTERIA IN THE AIR

The number of bacteria in the air ordinarily has a direct relation to the amount of dust; in fact, many of the bacteria in the air are attached to dust particles. Bacteria in the air are commonly considered as one kind of dust, but on account of their significance they are given separate consideration.

Bacteria are not found everywhere in the air; uninhabited places are quite free and the number diminishes as we ascend.

Bacteria do not multiply in the air; in fact, most of them soon die, especially when exposed in dry air to sunshine. For the most part, the bacteria in the air belong to the harmless varieties, although the number and kind vary greatly with circumstances. They come chiefly from the soil and are carried into the air by the wind and traffic movements; that is, bacteria in the air are derived from practically the same sources as dust. The dangerous bacteria in the air, however, come directly or indirectly from man and some of the lower animals.

The number of bacteria differs greatly with the local conditions. There are more in the air of towns than in the open country; few in high mountains, desert places, or at sea; more in windy weather than calm air; more indoors than in outside air; more in dry air than in moist air; more before than after rain. The air of badly ventilated rooms, especially if not kept clean, contains very many bacteria, and more when occupied, as the movements of the occupants stir up the dust.

Miquel of the Observatory of Montsouris studied the number of bacteria in the air of various localities. He found about 150 per cubic foot in the air of Paris, but only 6 after rain; on the top of the Pantheon he found 1½; in the streets about 12 per cubic foot; in a neglected hospital 3,170; in a gram of laboratory dust 75,000, and in a gram of house dust 2,100,000.

Flügge considers that on the average there are about one hundred

microorganisms to a cubic meter of city air—an average evidently below that of Paris.

Dr. Jean Binot did not find a single bacterium in 100 liters of outside air taken at the summit of Mont Blanc; and he found a progressive decrease in the number as the height increased. Thus, he found:

At Montanvert	49
At the Mer de Glace	23
At the Place de l'Aiguille	14
At the Grand Malet	8
At the Grand Plateau	6
On the summit	0

Again, Graham Smith found at the top of the Clock Tower of the Houses of Parliament in London only one-third of the number at ground level.

Pasteur, in experiments that will ever remain classic, exposed organic infusions in flasks to the air of various places, and used the results thus obtained to prove the presence or absence of bacteria in the air and to dispel the illusion of spontaneous generation. Of 20 such flasks exposed to the air of the Mer de Glace 19 showed no contamination. About the same time (1875) Tyndall exposed 27 flasks containing an infusion to the air of the Aletsch glacier (8,000 feet); none showed putrefaction, while 90 per cent. of the flasks opened in a hayloft were "smitten."

It is estimated that a person living in London breathes about 300,000 microbes in the inspired air each day.

The expired air, during normal respirations, is practically bacteria-free, no matter how many may be contained in the inspired air. The moist mucous membranes of the upper respiratory passages act as a bacterial trap. When the expired air contains bacteria it is only as a result of coughing, sneezing, talking, or other forced expiratory efforts (see Droplet Infection).

The harmful bacteria in the air and the danger of contracting disease through air-borne infection are considered in the next section.

Method for Determining Bacteria in the Air.—A rough idea of the bacterial population of the air may be obtained by exposing suitable culture media in Petri plates for various periods of time, and counting the colonies which develop from the germs falling upon them.

A large number of different devices have been described for a more accurate determination of the number of bacteria in the air. These are all adaptations of three general methods: (1) filtration of air; (2) bubbling air through some liquid medium; (3) precipitating the bacteria from a given volume of air. Each of these methods can be made to give fairly satisfactory results in the hands of competent workers,

but the Committee of the American Public Health Association recommend the following method of Petri on account of its simplicity and general applicability.

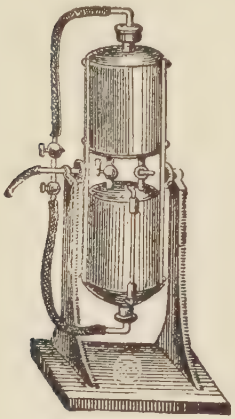


FIG. 87.—MAGNUS ASPIRATOR.

FILTRATION METHOD OF PETRI.—The filter tubes are glass tubes 1½ cm. in diameter and 10 cm. long. In the end of each is placed a perforated cork stopper, through which a glass tube 6 mm. in diameter is passed. The filtering material consists of sand which has been passed through a 100-mesh sieve. The sand in the filter tube is 1 cm. deep and supported by a layer of bolting cloth covering the cork. Two filter tubes are connected in tandem, and a measured volume of air, 10 liters or more, is drawn through at a constant rate by suction. The suction is applied by means of an aspirator of known volume, preferably one of the double or continuous type. Either the Magnus aspirator (Fig. 87) or the double aspirator (Fig. 88) are suitable for this purpose. Before using a pair of filter tubes a test for possible leakage is made by placing the thumb over the cotton stopper and applying the aspirator; if the suction is weak or absent the corks must be tightened or the tubes discarded. All corks should be tightened and connections wired and the apparatus sterilized before using the filters. The collection of the sample should take from 1 to 2 minutes per liter.

FIG. 88.—DOUBLE ASPIRATOR.

After filtering a definite volume through the tubes the sand is poured into 10 c. c. of sterile water, thoroughly shaken, and aliquot portions plated in ordinary nutrient agar, all plates being made in duplicate. The plates are incubated at room temperature for five days, when final counts are made.

RETTGER'S METHOD.—A new and improved method of enumerating air bacteria has just been described by Rettger,[1] which commends itself as the best method yet devised. The method consists of bubbling a given quantity of air through salt solution. The bacteria in the air are trapped in the salt solution, which may then be planted in the usual way and the number of colonies counted.

Air and Infection.—The air was long regarded as the vehicle and even the source of the communicable diseases. Theories, such as noxious effluvia, poisonous emanations, and infectious miasmata, gave way with the advent of bacteriology. When the early classical researches

[1] *Jour. of Med. Res.*, June, 1910, XXII, 3, pp. 461-468.

of Pasteur, Tyndall, and others showed that bacteria exist in the air almost everywhere in greater or in lesser numbers, the conclusion was jumped at that the air must be particularly dangerous. Within recent years, however, we have learned that the air is not very much to be feared on account of the bacteria it may carry, except under certain occasional circumstances. This change in our views during recent times is nowhere better illustrated than in the relation of the air to surgery. During the early days of antiseptic surgery so much fear was entertained for the bacteria in the air that Lister attempted to neutralize the danger with carbolic sprays and other means; now the surgeon pays little heed to the air of a well-kept operating room. Instead he ties several layers of sterile gauze over his mouth and nostrils and over his head to guard against particles falling from these sources.

It was one of the great surprises when bacteriologists demonstrated that the expired breath under normal conditions of respiration is sterile.

At one time many, if not most, of the contagious diseases were believed to be air-borne; many observations are on record purporting to prove that contagium may be carried long distances through the air. With the increase of our knowledge concerning the modes of transmission of infection the list of air-borne diseases has steadily dwindled. The theory is reluctantly given up, for it is the easiest method of explaining the spread of the readily communicable diseases. There are only two diseases of man, viz., smallpox and measles, which may possibly be air-borne, in the sense that this term is generally used. Both these diseases are so readily communicable that the virus seems to be "volatile"; it is assumed that the active principle is contained in the expired breath; however, there is no proof of this assumption, and some evidence to the contrary. Further, it is noteworthy that we are still ignorant of the causes and the precise mode of entrance of the contagium in both measles and smallpox. Even in these two diseases the radius of danger is much more limited than was once supposed to be the case.

The more the transmission of the communicable diseases is studied the less the air is implicated. The fact that malaria (bad air), yellow fever, and other diseases are conveyed by mosquitoes has robbed the air itself of false accusations, and given a death blow to miasms, effluvia, and intangible theories. Pettenkoffer insisted that the air became contaminated with poisons that were generated in a polluted soil, and he believed that these emanations were responsible in part for typhoid fever and cholera. Some association between soil, air, and disease still persists in both medical and lay minds, but with a more precise knowledge of the causes and modes of transmission of infections, such as typhoid fever and cholera, the air becomes a negligible factor. Out-of-door air contains relatively few bacteria; further, the

dilution is enormous. Most microorganisms pathogenic for man soon die when dried or when exposed to sunlight. Whatever danger, then, resides in the air, so far as living principles of disease are concerned, is found rather in indoor air, and especially in the air of badly ventilated, dusty, and crowded places. Here the danger may be either from the bacteria-laden dust or from droplet infection. In a crowded and stuffy street car, in a poorly ventilated office, or in a closed, close sickroom it would be very easy for the microorganisms of diphtheria, scarlet fever, whooping-cough, measles, pneumonia, influenza, common colds, tuberculosis, pneumonic form of plague, and other infections contained in the secretions from the nose and mouth to be held in the air in sufficient numbers so that exposed persons may contract the disease. This probably occurs more frequently than we are at present inclined to admit.

The radius of danger through droplet infection is quite limited It is difficult to conceive that infection may be carried long distances in the air and still be dangerous. My own experience indicates that there is practically no hazard in establishing a hospital for contagious diseases upon the high road or even in a thickly inhabited part of the city. In fact, the communicable diseases are not conveyed from ward to ward or even from bed to bed in well-managed hospitals.

Chapin states that many contagious hospitals have been maintained for years with no increase of the disease in the vicinity, as, for instance, at Boston and Providence, R. I. At the Kingston Avenue Hospital in Brooklyn various diseases, as smallpox, measles, scarlet fever, and diphtheria, are treated in wards only a few feet apart, with no evidence of aerial transference. At North Brother's Island the tuberculosis ward is only about 25 feet from the diphtheria ward, but the tuberculous patients do not contract diphtheria. A number of hospitals for communicable diseases have recently been built with entire disregard of aerial infection. At the hospital of the Pasteur Institute, Paris, the patients are each cared for in a separate ward opening into a common hall. The same nurses go from case to case. In 2½ years after it was opened in 1900 there were treated 2,000 persons, of whom 524 had smallpox, 443 diphtheria, 126 measles, 163 erysipelas, 92 scarlet fever, and 166 non-diphtheritic sore throat. The only evidence of the transfer of infection was the development of four cases of smallpox and two of erysipelas. In the Hôpital des Infants Malades in Paris the beds, instead of being in separate rooms, are separated by partitions. Of 5,017 cases there were only 7 cross infections, 6 of measles and 1 of diphtheria. These were attributed to lapses in aseptic precautions. Dr. Moizard thinks that this experience proves that even measles is not air-borne. Dr. Grancher in another Paris hospital has two wards in which there are no partitions, but only wire screens around the beds,

simply as a reminder for the nurses. He also insists that measles is probably not an air-borne disease, and that adjacent patients do not necessarily infect one another. At various English hospitals similar methods have been tried with success.[1]

While the air plays a minor rôle in the spread of the infections, bad air plays an important part in reducing vitality and predisposing to disease. This will be discussed presently.

POISONOUS GASES IN THE AIR

Some of the poisonous gases of the air come from natural sources, as marshes, mines, or decomposing organic matter, but those that concern the sanitarian particularly are the gases which arise from the works of man. These gases are carbon monoxid, ammoniacal vapors, hydrochloric acid, carbon bisulphid, carburetted hydrogen, hydrogen sulphid, etc.

Carbon Monoxid.—Carbon monoxid (CO) is a frequent and serious cause of chronic ill health or acute poisoning. The carbon monoxid in the air comes principally from illuminating gas, but it is also found about lime kilns and where coke fires and brasiers are used in confined spaces; also from iron and copper furnaces, from coal fires, and other sources. Air containing 0.4 per cent. of carbon monoxid may, in one hour, prove fatal. In higher concentration a person may be overcome at once and death soon ensues. Chronic poisoning with smaller amounts may lead to anemia, depression, and other symptoms.

Carbon monoxid or carbonic oxid is a colorless and practically odorless gas; it burns with a pale blue flame. Its poisonous action depends upon the fact that it combines with the hemoglobin of the red blood corpuscles to form carbon monoxid hemoglobin. This is a stable compound which, therefore, prevents the hemoglobin giving up its oxygen to the tissues. When present in only small amounts in the atmosphere the effects of CO may be compensated for by a polycythemia—an increased number of corpuscles taking the place of those disabled.

Carbon monoxid may be found in the air of inhabited rooms as a result of leaking gas fixtures, imperfect combustion of illuminating gas, and defects in apparatus fed by coal. Burning charcoal yields CO in great abundance, and it is also given off from red-hot cast-iron stoves, and, finally, as a result of incomplete combustion of coal in furnaces and ranges.

One of the most common sources of carbon monoxid in the household is from illuminating gas. Illuminating gas may pass from a broken gas main through the soil into the cellar and thence permeate a dwelling; this is aided by the suction and pumping action of the heating apparatus in the cellar. In passing through the soil illuminat-

[1] Chapin: *Jour. Am. Med. Ass'n.*, Dec. 12, 1908, Vol. LI, pp. 2,048-2,051.

ing gas may be robbed of its characteristic odor, thus rendering it so much more dangerous because not perceived. The danger from this source is further increased in the winter time and in cities with asphaltum and concrete pavements, because under these circumstances the escape of gas into the air is hindered and the chance of more of it reaching the house through the cellar is favored. An occasional source of CO in the air of houses is through hot-water heaters, using illuminating gas as fuel. The soot gradually collects in these devices and may become incandescent, thus furnishing ideal conditions for the production of carbon monoxid. In the arts CO is formed by passing water vapor over incandescent carbon. I know of one case in Washington where CO from a water heater collected in a kitchenette in such concentration that three persons were overcome upon entering the room and died.[1]

Illuminating Gas.—Illuminating gas may be harmful either from the products of its combustion or, more so, when the unconsumed gas escapes in the household. The two principal illuminating gases used are coal gas and water gas. The poisonous effects of both are due mainly to the carbon monoxid which they contain.

Coal gas is made by the destructive distillation of coal. It contains hydrogen, marsh gas, and carbon monoxid, occasionally also ethene, acetylene, and carbon dioxid. A cubic foot of coal gas completely burned gives to the atmosphere about one-half a cubic foot of CO_2 and about 1.34 cubic feet of water vapor. An ordinary gas jet burns about 6 cubic feet of gas per hour, and thus produces about 3 cubic feet of CO_2.

Water gas is made by blowing a current of steam through incandescent coke or coal. The water is decomposed into hydrogen and oxygen. The hydrogen passes on and the oxygen unites with the carbon to form carbon monoxid. Water gas so produced burns only with a pale blue flame. It is, therefore, enriched in a carburetor with vaporized petroleum; this furnishes the hydrocarbons necessary to give a luminous flame. Water gas contains about 30 per cent. of carbon monoxid.

The effect of these carbonaceous illuminants is to elevate the temperature and increase the moisture of a room. They also add carbon monoxid, carbon dioxid, nitric and nitrous acid, compounds of ammonia and sulphur, marsh gas, carbon particles (soot), acids of the fatty group in small but variable amounts. The following instructive table gives the comparative candle power and also the gases and heat produced by the usual forms of illuminants:

[1] For methods for determining carbon monoxid and other gases in the air, see: Haldane, J. S.: "Methods of Air Analysis." J. P. Lippincott and Co., 1912.

	Quantity Consumed	Candle-power	Oxygen Removed	Carbon Dioxid Produced	Moisture Produced	Heat Calories Produced	Vitiation Equal to Adults
	grains		cu. ft.	cu. ft.	cu. ft.		
Tallow candles	2,200	16	10.7	7.3	8.2	1,400	12.0
Sperm candles	1,740	16	9.6	6.5	6.5	1,137	11.0
Paraffin oil lamp	992	16	6.2	4.5	3.5	1,030	7.5
Kerosene oil lamp	909	16	5.9	4.1	3.3	1,030	7.0
	cu. ft.						
Coal gas, No. 5 Batswing burner	5.5	16	6.5	2.8	7.3	1,194	5.0
Coal gas, Argand burner	4.8	16	5.8	2.6	6.4	1,240	4.3
Coal gas, regenerative burner	3.2	32	3.6	1.7	4.2	760	2.8
Coal gas, Welsbach incandescent	3.5	50	4.1	1.8	4.7	763	3.0
	lb. coal						
Electric incandescent light	0.3	16	0.0	0.0	0.0	37	0.0

Most coal contains sulphur, which appears in coal gas as sulphuric acid, which is irritating and poisonous. Most of the sulphur compounds in coal gas are removed by processes of purification during manufacture, but, owing to the difficulty of complete removal, 20 grains of sulphur in every hundred cubic feet are generally allowed by law. The sulphur restrictions have recently, but unwisely, been removed in England. In Massachusetts the legal limit has been raised to 30 grains per hundred cubic feet. These changes were brought about by the claims of gas companies that it is much more difficult than formerly to procure coals low in sulphur, so that the processes for the removal of the sulphur have become costly and burdensome.

Illuminating gas is required by law, in Massachusetts and in many other places, to be free from ammonia as well as sulphuretted hydrogen, but this is more because of injury to fixtures than because of danger to health.

Water gas is cheaper than coal gas, and is, therefore, preferred by gas companies. Usually a mixture of the two gases is supplied. Experience shows that if water gas is properly diluted with coal gas the danger is greatly lessened. Illuminating gas containing 6 per cent. of carbon monoxid is not hazardous. Most cities limit the amount to 10 per cent. In 1890 the 10 per cent. statute was repealed in Massachusetts, and it is since then that the marked increase in illuminating gas poisoning has occurred. There were 1,231 deaths caused by illuminating gas in Massachusetts during the years 1886 to 1909. About one-half of these deaths were suicidal. This only represents the fatalities, and does not take into account the many cases of chronic poisoning which occur in the home and in the industries where much illuminating gas is used.

Sedgwick and Schneider[1] state that the death rate from poisoning

[1] *Jour. of Infect. Dis.*, Vol. IX, No. 3, 1911.

by illuminating gas in Massachusetts and Rhode Island has become nearly equal to that of scarlet fever or measles.

Gas pipes in a dwelling should be tested from time to time with a pressure gage, and minor leaks from faulty stopcocks, from "rubber" tubing used for droplights, etc., should be carefully searched for and corrected. A flaring gas burner is not only wasteful, since it implies the escape of unburned gas, but is also harmful to health. A gas jet should burn steadily without jumping and flaring.

Other Gases in the Air.—Ammoniacal Vapors.—Ammoniacal vapors irritate the conjunctiva, but have no other evident effect on health in the amounts ordinarily found in the air.

Hydrochloric Acid Vapors.—Hydrochloric acid vapors in large quantities are very irritating to the conjunctiva and respiratory mucous membranes. In the alkali manufactures they are sometimes poured into the air in sufficient quantity to destroy vegetation. When in sufficient concentration they may induce bronchitis, pneumonia, and even destruction of lung tissue, as well as inflammation of the eyes.

Carbon Bisulphid.—Carbon bisulphid is given off in the vulcanizing of India rubber. It produces headache, vertigo, pains in the limbs, formication, sleeplessness, nervous depression, and loss of appetite; sometimes deafness, dyspnea, cough, febrile attacks, and even paraplegia. The effects seem due to a direct anesthetic action on the nervous tissue.

Carburetted Hydrogen.—As much as 200 to 300 volumes per 1,000 of carburetted hydrogen may be breathed for a short time. Above this amount it produces headache, vomiting, convulsions, stertor, dilated pupil, etc. Breathed in small quantities, as it constantly is by some miners, it is not known to produce any bad effects, although such may be the case.

Hydrogen Sulphid.—The susceptibility of man to this gas varies. Its dangerous nature is fully recognized in all chemical laboratories. The effects of small amounts are not well understood. Thackrah could find no bad effects. On the other hand, Hirt believed it produced chronic poisoning. The symptoms are chiefly weakness, depression, anorexia, slow pulse, furred tongue, and marked pallor.

Sulphur Dioxid.—Sulphur dioxid is extremely irritating and causes bronchitis. Those exposed to the fumes in the bleaching of cotton and worsted goods are frequently sallow and anemic.

SEWER GAS

Sewer gas, once a hygienic bugaboo, is now not seriously regarded by sanitarians. Sewer gas became the residual legatee of Murchinson's pythogenic theory, namely, that typhoid fever was "produced by emana-

tions from decaying organic matter." Sewer "gas" is nothing more or less than air containing the volatile products of organic decay coming from sewers and drains. Sewer gas is a variable mixture, both as to composition and concentration. Some of these gases are more or less poisonous, but not in the great dilution ordinarily found in sewer air. As a matter of fact, the air of sewers is ordinarily freer of dust and bacteria than the corresponding outside air, although it may be a little higher in carbon dioxid—10 to 30 volumes per 10,000. It is absurd to regard sewer gas as the cause of diphtheria, typhoid fever, scarlet fever, and other communicable diseases. So far as unpleasant odors are concerned, they are more apt to come from defective drains or unclean and unventilated house plumbing than from a well-constructed sewer. Workmen employed in sewers and about sewage ordinarily remain hale and healthy.

Bacteria in Sewer Air.—When it was found that there are no dangerous volatile poisons in sewer air attention was focused upon the bacteria; however, Nägeli as long ago as 1877 showed that putrescent liquids kept in the same sealed vessel for over two years did not infect each other. Sir Edward Frankland then showed that lithium carbonate in solution did not contaminate the air, but that when effervescence was produced the breaking of the bubbles on the surface of the liquid carried the lithium a distance of 21 feet up a vertical tube. The inference was that sewage through fermentation or splashing may send bacteria into the air. Pumpelly in 1881 and others since have shown that bacteria are not given off from a liquid if the surface remains unbroken, even though the air may blow over it. In 1893 Miquel began a monumental work upon bacteria of the air. He made routine observations at the Montsouris Observatory, and for four years compared the bacteria in the air of a Paris street with the air of sewers. He found sewer air relatively pure from a bacteriological standpoint. Carnelly and Haldane in 1877 found fewer bacteria in the sewers under the House of Parliament and other places than in the air of adjacent streets. The number of bacteria was largest in the best-ventilated sewers, because these brought the street bacteria along with them. Abbott in 1894 showed that cultures of *B. prodigiosus* are not carried over in bubbles produced by natural fermentation (yeast in a carbohydrate medium), but may be carried a short distance by blowing air at considerable velocity through the culture. He concluded that the danger of bacteria being transmitted from sewage into the air under ordinary circumstances is practically negligible. In 1907 Horricks revived this question by placing *B. prodigiosus* in the water-closets of a large military hospital in Gibraltar, and recovering them on plates suspended on top of the soil pipes and in manhole openings. His work gave countenance to the views of a number of English sanitarians, who maintain the reality

of the danger from this source. Winslow repeated Horrick's experiments in 1909, using the ordinary sewage of Boston, and by using quantitative methods threw a different light upon Horrick's conclusions. He found that a vigorous foaming produced very slight bacterial infection of the air—only five prodigiosus colonies in 30 liters of air. Further, the infection always remained localized. Generally he found the air of house drains singularly free from bacteria. It, therefore, seems theoretically possible, but very improbable, that infection may take place in this way. Practically the question seems to have little importance. Thus, out of a series of examinations of plumbing systems in actual use, Winslow found intestinal bacteria only four times in 200 liters of air, and these directly at the point of local splashing.

If there is any danger of sewage bacteria coming into our houses, it is rather that they are dragged in by rats, roaches, water bugs, and other vermin that use sewers and drains as highways.

Ventilation of Sewers.—Sewers cannot be constructed airtight on account of the numerous openings into them. The tension of the air in sewers is generally not very different from that of the atmosphere outside. The movement of the air is generally in the direction of the flow of the current. The simplest plan of ventilation is by means of a shaft from the top of the sewer to the surface of the street or road above, where the opening of the shaft should be covered by an iron grating. These openings are usually placed at intervals of 100 yards or so. This system, which is in common use, has been much criticized, mainly on account of the fact that the objectionable gases are discharged more or less immediately under the noses of passers-by. To meet this objection it has been proposed, and actually come about in some places, to locate tall iron shafts at suitable intervals to permit the discharge of air and gases at a level well above the roofs of houses. As a matter of fact, if sewers are well constructed, have sufficient fall and flow of water, there will be no accumulation of foul gases. One of the main causes of decomposition is due to dead ends. These should not be tolerated by the engineer in charge of the sewage department. Recently an agitation has been started to solve this question of sewage ventilation by advocating the abolition of the intercepting traps on the house drains between the sewer and the house, thus converting every house drain and every soil pipe into so many sewer ventilators. There are many objections to this plan, as it would destroy the drain isolation between each house, which is now possible from the sewer, and from the neighboring houses of the district.

CHAPTER V

FRESH AND VITIATED AIR

THE BENEFITS OF FRESH AIR

Fresh air is nature's tonic. It stimulates digestion, promotes assimilation, improves metabolism, strengthens the nervous system, and increases our resistance against some diseases. It is a common experience that fresh air gives us a general feeling of well-being. Much of the benefit of an outdoor life comes also from the exercise, diversion, sunshine, and other factors. The stimulating effect of outdoor air varies considerably with the temperature and movements of the air. Cold air is especially stimulating, and much of the good of sleeping out of doors is perhaps secondarily due to the tonic action of cold. Sleeping out of doors or with open windows atones for much bad air during the daytime. However, the good results of fresh air may be neutralized by undue exposure to cold, especially in the young, the aged, and the feeble—or even in robust individuals not properly protected.

"We may write and talk as much as we please about the horrors of bad air and the importance of fresh air, but we should never induce people to sit in cold drafts and shiver for the sake of pure air, and, in fact, we would not want to do it ourselves" (Macfie). Extremes in this as in all matters hygienic are to be avoided. It is important that those who sleep out of doors or sit out, should be warmly clad and sufficiently fed.

THE EFFECTS OF VITIATED AIR

The effects produced by an atmosphere vitiated by the breath and other exhalations from human beings may be divided into acute and chronic. The acute effects are usually lassitude, headache, vertigo, nausea, vomiting, and even collapse. In extreme cases death may ensue. The chronic effects, so far as is known, include anemia, debility, and disturbances of digestion. Prolonged exposure to vitiated atmospheres also influences nutrition and metabolism, depresses vitality, and lowers the resistance to certain infections, especially to the pyogenic

cocci, the tubercle bacillus, the pneumococcus, and to the microorganisms causing common colds. It is often difficult, especially in the poorer classes, to know how much is due to bad air and how much to poor food, overwork, loss of sleep and rest, worry, and other inflictions of poverty. There is plenty of evidence to show that men living in insufficiently ventilated barracks and other habitations have a high death rate. The lower animals under like conditions in crowded and poorly ventilated stables also have a high mortality. The statistical evidence of the English Barrack and Hospital Commission, published in 1861, shows that men living a considerable portion of their time in badly ventilated rooms have a higher death rate than those having well-ventilated rooms, other conditions being about the same. The high morbidity and mortality in crowded places are due, in part at least, to the favorable conditions for the spread of the communicable diseases, and must not be laid entirely to the effects of vitiated atmospheres.

Some Extreme Cases.—The acute and fatal effects caused by breathing a seriously vitiated atmosphere, under unusually severe conditions, are well illustrated by the three following instances:

After the battle of Austerlitz 300 Austrian prisoners were shut into a prison in a small cellar, and 260 were killed by the impure air in a few hours.

In the tragedy known as the Black Hole of Calcutta, the military prison of Fort William, January 18, 1756, 146 adults were shut into a room only 18 feet square and with but two small windows on one side to ventilate it. They were shut in at 8 P. M., and within an hour some were dead, and when the door was opened at 6.20 next morning only 23 were found to be alive. One of the survivors gives the following description of the horrors of the night: "At this period so strong a flavor came from the prison that I was not able to turn my head that way for more than a few seconds at a time. Everybody except those at the windows now grew outrageous and many delirious. By eleven o'clock greater numbers were dead or dying, and those living were in an outrageous delirium and others quite ungovernable. A steam now arose from the living and the dead, which most awfully affected those who were still alive. At six o'clock next morning it came to the ears of the Indian governor the havoc death had made in this fearful place, and he ordered their release. At 6.20 there came out of this living grave 23 *half-dead creatures*, being all that remained of the 146 souls who had entered the Black Hole prison, and these were in such a condition that it seemed very doubtful whether they would see the morning of another day. Many of the survivors developed putrid fever and boils. The remaining 23 were poisoned by exhalations from their own lungs and bodies."

An almost equally terrible tragedy took place on the steamer *Londonderry*, going between Sligo and Liverpool. The tragedy is thus described by G. Henry Lewes ("Physiology of Common Life"):

"On Friday, December 2, 1848, she left for Liverpool with two hundred passengers on board, mostly emigrants. Stormy weather came on, and the captain ordered every one below. The cabin for the steerage passengers was only 18 feet long, 11 feet wide, and 7 feet high. Into this small space the passengers were crowded; they would only have suffered inconvenience if the hatches had been left open; but the captain ordered these to be closed, and—for some reason not explained—he ordered a tarpaulin to be thrown over the entrance to the cabin and fastened down. The wretched passengers were now condemned to breathe over and over again the same air. This soon became intolerable. Then occurred a horrible scene of frenzy and violence, amid the groans of the expiring and the curses of the more robust; this was stopped only by one of the men contriving to force his way on deck, and to alarm the mate, who was called to a fearful spectacle: seventy-two were already dead, and many were dying; their bodies were convulsed, the blood starting from their eyes, nostrils, and ears."

The foregoing instances are exceptional, and for practical purposes may be regarded simply as the results of suffocation. The usual conditions never approach such extremes, but are, nevertheless, important, for they may be serious. We must first consider the question why an atmosphere vitiated by the presence of human beings produces ill effects.

Three explanations have been offered: (1) increase of carbon dioxid and diminution of oxygen; (2) poisons in the expired breath; (3) physical changes of the air. Each of these explanations will be considered separately.

The Effects of Increased Carbon Dioxid and Diminished Oxygen.—According to the older theories, the sensations of discomfort, arising in inclosed places, had their origin either in an excess of carbon dioxid or an insufficiency of oxygen. Thus, in the early experiments of Claude Bernard (1857) animals were confined in atmospheric air and in mixtures both richer and poorer in oxygen than atmospheric air. He explained the poisonous effects of carbonic acid when respired to be due to the fact that it deprived the animal of oxygen. Similar results were reported by Valentin and by Paul Bert. Richardson in 1860-61 found that a temperature much higher or lower than 20° C. had the effect of shortening very considerably the lives of animals confined in an unventilated jar. Pettenkoffer in 1860-63 cast the first serious doubt on the correctness of these theories. He believed that the symptoms observed in crowded, ill-ventilated places were not produced by the excess of carbonic acid nor by a decrease in the proportion of oxygen in the air. He further did not believe that the impure air of dwellings

was directly capable of originating specific diseases, or that it was really a poison in the ordinary sense of the term, but that it diminished the resistance on the part of those continually breathing such air.

Hermans[1] showed that an atmosphere containing only 15 per cent. of oxygen and as much as 2 to 4 per cent. of carbon dioxid may not be harmful. On removing the carbon dioxid there was no great discomfort, even when the oxygen was reduced to 10 per cent. The air of certain breweries examined by Lehmann[2] contained 1.5 to 2.5 per cent. of carbon dioxid, and men worked continuously in this for years without any ill effects. The CO_2 occasionally rose to 6 and even 10 per cent., but then was liable to produce temporary intoxication. It is now generally admitted, upon the testimony of numerous experimenters, that an atmosphere containing as much as 3 per cent. of carbon dioxid and as little as 15 per cent. of oxygen has no toxic effects and produces no disturbing symptoms. In the most poorly ventilated rooms the carbon dioxid never reaches this amount, especially when produced by respiration alone. It is unusual to find 0.5 per cent. It is, therefore, plain that we must look to other causes for the effects of vitiated air.

Poisons in the Expired Breath.—In 1863 Hammond believed he demonstrated the presence of organic matter, because, when vitiated air is passed through potassium permanganate, it decolorizes that strong oxidizing agent. Hammond confined a mouse under a jar in which the CO_2 was taken up by baryta water as fast as it was formed and the moisture absorbed by calcium chlorid. Nevertheless, the mouse died in 40 minutes. The observation was repeated a number of times, and death ensued invariably in less than one hour. Brown-Séquard and D'Arsonval in 1888-9 claimed to be the first to demonstrate poisonous bodies in the expired breath. They condensed the moisture in the exhaled breath, which was injected into the veins of rabbits. Death usually took place in a few days, sometimes in a few weeks. They believed from this that they had discovered a volatile organic poison of the nature of an alkaloid, similar to Brieger's ptomains. These experiments were repeated with variable results, but in 1889 they reported ingenious experiments in which they obtained additional evidence in support of their former statements. Rabbits were confined in a series of jars connected with rubber tubing, permitting a constant current of air to be passed. The animal in the last jar received the air from the lungs of the animals in the other jars. This animal died after an interval of some hours, and the animal in the next jar was the next to die. The first and second animals usually remained

[1] Hermans: "Ausschaltung organischer Substanzen durch den Menschen," *Archiv f. Hyg.*, 1883, I, 1.

[2] Lehmann: "Untersuchung über die langdauernde Wirkung mittlerer Kohlensäuredosen auf den Menschen," *Arch. f. Hyg.*, 1899, XXXIV, 335.

alive. When absorption tubes containing concentrated sulphuric acid were placed between the last two jars, the animal in the last jar remained alive while the one in the jar just before was the first to die. These results confirmed their belief in the existence of a volatile poison absorbed by the sulphuric acid. Haldane and Smith repeated the experiments of Brown-Séquard and D'Arsonval, using five bottled mice. They continued the exposure for 53 hours without ill effects to the mice. Beu in 1893 also repeated these experiments, and came to the conclusion that acute poisoning through the organic matters contained in the expired air was not possible, and that the death of the animals was due to changes of temperature and accumulation of moisture in the jars. Rauer in 1893, also Lübberd and Peters, concluded from similar experiments that there are no organic poisons in the expired air. In fact, Merkel stands almost the only sponsor for the correctness of the conclusions of Brown-Séquard and D'Arsonval, and with some slight changes of technique he was unable to get uniform results.

Lehmann and Jessen in 1890 collected from 15 to 20 c. c. of condensed fluid per hour from the breath of a person exhaling through a glass spiral laid in ice. This fluid was always clear, odorless, neutral in reaction, and contained slight traces of ammonia with good teeth; more with poor teeth. Inoculation of this condensed fluid into animals gave negative results. Many other experimenters, including von Hoffman-Wellenhof, Lehmann and Jensen, Haldane and Smith, Billings, Weir Mitchell, and Bergey, have shown that the fluid condensed from the breath is no more toxic than distilled water when injected into animals. This has strengthened the general belief that poisonous bodies are not present.

In 1894 Brown-Séquard and Davis reported further experiments in which they inoculated over one hundred animals with the condensed fluid of respiration, and not only confirmed their former statements, but were unable to understand the failure of other experimenters, and emphatically reaffirmed that the breath contains a volatile poison and that the death of animals under experimental conditions is not due to an excess of carbon dioxid nor a deficiency of oxygen. These experiments were repeated by Billings, Mitchell, and Bergey [1] in 1895, who came to the conclusion that the ill effects of vitiated atmosphere depend almost entirely upon increased temperature and moisture, and not on an excess of carbon dioxid or bacteria or dust of any kind. They admit that the cause of the musty odor in unventilated rooms is unknown.

In addition to reducing potassium permanganate, it has been shown that the breath contains traces of ammonia and traces of hydrochloric acid. These have their origin in decaying teeth and decomposing par-

[1] Published by Smithsonian Institution, 1895. Contains a summary of the literature to date, with references to authorities.

ticles of food or other putrefactive or pathological changes occurring in the upper respiratory passages. The ammonia and hydrochloric acid exist in such small quantities that they have no practical bearing upon the question under consideration.

Weichardt [1] calls attention to the fact that putrefactive processes go on in the excretory products of the respiratory tract, especially in older persons. He states that the bronchial mucus of corpses contains a poison resembling kenotoxin (the toxin of fatigue). When injected into laboratory animals it produces a lowering of temperature, a slowing of respiration, and death. According to Weichardt, fluids condensed from the expired air and then concentrated, when injected into mice, produce like results. This investigator also evaporated some of the condensed moisture from the expired breath and obtained a weighable residue (9 milligrams from 10 c. c.). This he regards as partly organic matter. As further proof that the organic matter in the expired breath is active, he obtained from the expired breath of a tired old man the condensed fluid which he then concentrated. This concentrated fluid has a distinct inhibitory effect upon the oxidizing power of the ferments in blood, as shown by the guaiac indicator. Also by means of the epiphanin reaction Weichardt considers that he has demonstrated protein-split products in the vitiated air of a room. He concludes that substances having such important biological power should not be longer overlooked. These results lack confirmation, and the methods are open to criticism.

Rosenau and Amoss [2] demonstrated the presence of protein matter in the expired breath through the reaction of anaphylaxis. The first injection into guinea pigs of the fluid, obtained by condensing the moisture of expiration, is harmless, but the animals become sensitized, so that they react to an injection of human blood serum after an interval of several weeks. Of 99 guinea pigs tested 26 responded definitely; in 4 of the animals the reaction was so severe that death ensued from anaphylactic shock. This plainly indicates that protein matter, specific in nature, is present in the expired breath. It is inferred that the protein matter probably comes from the blood. These experiments, however, do not prove that these substances are poisonous.

Further researches have shown that guinea pigs may be sensitized by exposing them to the expired breath of dogs. That is, the protein matter thrown off in the dog's breath may be inspired and absorbed by the guinea pigs. This indicates that organic matters in the expired breath may have a physiological significance, but we have no proof that

[1] Weichardt: "Ueber Eiweifsspaltprodukte in der Ausatemluft," *Arch. f. Hyg.*, 1911, 74 Bd., Heft 5.

[2] "Organic Matters in the Expired Breath," *Jour. of Med. Research,* Vol. XXV, No. 1, Sept., 1911, page 35.

they are poisonous in the sense in which that term is commonly employed. There is, therefore, no present proof that the expired breath contains a poisonous substance.

Physical Changes in the Air.—Owing to the failure of chemistry to demonstrate the cause of the ill effects produced by a vitiated atmosphere, attention has recently been focused upon the physical changes, such as the increase in temperature, increase in humidity, and the stillness of the air in a poorly ventilated room. Important experiments were carried out above five years ago in the Institute of Hygiene in Breslau by Heymann, Paul, and Erclentz. Flügge,[1] who was then the director of the institute, has admirably summarized and interpreted the results as follows:

Paul placed healthy individuals in a cabinet of 3 cubic meters' capacity, where they were kept for a variable time up to four hours, and until the carbon dioxid had risen to 100 or 150 parts in 10,000—an accumulation of gaseous excretion practically never developed under ordinary conditions. In these experiments no symptoms of illness or discomfort developed so long as the temperature and moisture were kept low. Tests of the psychic fatigue of these individuals by means of the esthesiometer and ergograph, or by means of computations, gave negative results throughout, under similar conditions of temperature and moisture. Tests in a crowded schoolroom were similarly negative. Erclentz made the same observations on diseased persons. Those suffering from emphysema, heart diseases, kidney diseases, etc., with the exception of a few peculiarly susceptible anemic and scrofulous school children, bore the highly vitiated air for hours without any evidence of bodily or mental depression.

The results were very different, however, when the temperature and moisture of the air of the cabinet were allowed to increase. At 80° F. with moderate humidity, or at from 70° to 73.5° F. with high humidity, practically all persons began to show depression, headache, dizziness, or a tendency to nausea. The susceptibility was not alike for all. School children reacted slightly and emphysematics slightly, while those with heart troubles were most susceptible. By means of certain objective signs of heat stagnation—the surface temperature of the forehead and the temperature and moisture of the clothed parts of the body—it was determined that subjective symptoms appeared only when the surface temperature reached a certain height. This was, for healthy people, 93° F. to 95° F. on the forehead; for the more susceptible and diseased, 89.5° to 91.5°; and with the moisture of the skin increased by 20 or 30 per cent. Under these conditions the normal dissipation

[1] Flügge: *Ztschr. f. Hyg.*, 1905, XLIX, 363. Crowder: *Archives of Internal Medicine*, Jan., 1911, Vol. VII, pp. 85-133. Contains an admirable summary and references to recent literature upon the subject.

of body heat is interfered with, and it is under these conditions that symptoms appear which are in every way similar to those developed in overfilled and "stuffy" rooms.

Now, when these people in the cabinet suffering from such symptoms were allowed to breathe the fresh outside air through a tube, such air being raised to the temperature and relative humidity of that within, it gave them no relief whatever; nor did the internal air produce any symptoms when breathed through a tube by one outside of the cabinet. But the symptoms of discomfort and illness experienced by the person within could be almost immediately relieved either by drying the air of the cabinet or by cooling it, or by putting it in rapid motion by means of a fan, without any chemical change being made in the air. The effect of these measures is simply by purely mechanical means to enable the body to throw off its heat more rapidly, and thereby all symptoms disappear; heat stagnation is the cause of the discomfort.

From the long series of experiments, carried out with great care as to all the details of observation and control, it is concluded that all of the symptoms arising in the so-called vitiated atmosphere of crowded rooms are dependent on heat stagnation in the body, and that the thermic conditions of the atmosphere, its moisture, and its stillness are responsible for the effects. To change any one of these elements is to change the rapidity of the loss of heat. If the change is such as to increase this loss, comfort is restored. It is also considered proved beyond any reasonable doubt, by their own as well as by previous research, that there is no gaseous excretion into the surrounding air, either from the breath or from other sources, deserving of the name of poison.

Angelici,[1] working independently at about the same time, concurs in these opinions; and Reichenbach and Heymann[2] later determined that objective evidence of heat stagnation in the body always precedes the development of subjective symptoms of discomfort under natural conditions, in the same way that it does under the artificial conditions of the cabinet.

Leonard Hill[3] of England also has confirmed these general results and conclusions. It should be noted that it is exceedingly difficult in these cabinet experiments to exclude the psychic factor.

[1] Angelici: Quoted by Reichenbach and Heymann, *Ztschr. f. Hyg.*, 1907, LVII, 23.

[2] Reichenbach and Heymann: "Untersuchungen über die Wirkungen klimatischer Factoren auf den Menschen," *Ztschr. f. Hyg.*, 1907, LVII, 23.

[3] Hill, Leonard, Rowland, R. A., and Walker, H. R.: "The Relative Influence of the Heat and Chemical Impurity of Close Air," London Hosp. Med. Col., *Journal of Physiology*, XLI, 1911.

SUMMARY

It is now perfectly plain that the ill effects resulting from a vitiated atmosphere are not due to an increase of carbon dioxid nor to a diminution in oxygen. Upon this point all are agreed. The general consensus of opinion also excludes poisonous bodies in the expired breath as a factor. On this point, however, the last word has not been said. The expired breath appears to contain protein bodies biologically active, which may have an influence upon health.

Sanitarians are satisfied, with the evidence presented, that most at least of the discomfort is due to physical changes only. If a normal heat interchange can be maintained between the body and the air the symptoms which are commonly attributed to poor ventilation do not develop. According to this view the vital element of the ventilation problem becomes that of regulating the temperature, moisture, and motion of the air. When the air is still we are surrounded by an "aerial envelope" with a temperature and moisture resembling the open air on a hot and humid day. The symptoms caused by crowd poisoning, such as oppression, malaise, headache, vertigo, nausea, vomiting, and even collapse, indeed resemble those of heat exhaustion.

Even those who look upon the physical changes in the air as the sole cause of the discomfort rather than the possibility of chemical changes admit that a certain amount of fresh air must be supplied. Flügge himself urges that life in the open should be more and more resorted to, but he would have the motive correctly understood, not that the chemical condition of inside air is harmful, but that it is the overheating of rooms that causes disturbances of health. Flügge states that one should go into the open not because he may breathe chemically purer air, but because its almost constant motion carries away the body heat and causes a beneficial stimulation of the skin and reflexly brings about a heightened cell activity that aids in the development of sturdy health. The chemistry of air and "crowd poisons" have little or no part to play in the explanation of outdoor benefits or of indoor discomforts. These are both dependent upon physical conditions, and their explanation rests with the physics of heat interchange between the body and its surrounding medium.

There is some danger in regarding the ill effects of poor ventilation as due to thermal and other physical factors alone. According to this theory it is only necessary to keep the temperature and moisture down and keep the air in motion; a closed office with an electric fan would take the place of any system of ventilation. There is already a clamor against the laws requiring fresh air in workrooms, based upon Flügge's views. This is a natural corollary of Flügge's views. If re-breathing

the same air is not hurtful, the ventilation of living rooms may be greatly simplified by simply keeping the physical conditions of the air within the limits of comfort. Furthermore, a great economy would be effected. It is, however, not scientific to insist that the chemical changes in a vitiated atmosphere may be disregarded, because we cannot at present demonstrate immediate relationship between cause and effect; neither is it safe to deny dogmatically the existence of injurious substances in a vitiated atmosphere simply because in the present state of our knowledge chemistry has failed to demonstrate them, and because most of the symptoms may be explained upon disturbances of thermic interchange.

Furthermore, most of the observations have been based upon short exposures; it is very probable that a decrease in mental and physical efficiency would result from a prolonged exposure to a vitiated atmosphere, even though it were kept dry and cool. The improvement in appetite, nerve vigor, blood quality, and muscular tone which follows open air treatment, even in the rich and well-fed, shows the paramount importance of fresh air.

CHAPTER VI

VENTILATION AND HEATING

VENTILATION

The problem of ventilation is apparently a very simple one; all that is required is to furnish a never-ending stream of fresh air from the inexhaustible supply without to replace that which is constantly being vitiated. To do this under the artificial conditions of house and factory life is often extremely difficult, and under certain circumstances practically impossible. Further, the problem of ventilation must take into account not only the quantity of air, but its physical condition, in order that the human machine may operate at the highest level of health and efficiency.

Ventilation must serve a number of purposes and comply with a number of conditions before it can be considered satisfactory: (1) it must bring pure air from without in order to dilute and remove the products of respiration, as well as other sources of vitiation; (2) it must maintain the air within the room at a proper temperature and humidity, and, further, must keep the air of the room in gentle and continuous motion; (3) it must remove the gases, odors, bacteria, dust, and other substances that contaminate the air of inclosed spaces; (4) it must dilute and remove the impurities produced by the burning of gas, candles, lamps, and other sources.

The purpose of ventilation is not to bring outdoor conditions indoors; the art of ventilation consists in adapting indoor conditions to indoor life. Indoor life is necessary in order to perform the delicate manipulations which cannot, as a rule, be effectively conducted outdoors. Indoor life, then, involves quiet and protection from sudden changes or extremes.

It is a simple mechanical problem to condition the air of an apartment. The ventilating engineer finds no difficulty in regulating the temperature and humidity within narrow limits, and in furnishing definite quantities of fresh, moving air. To maintain these conditions, however, the doors and windows must be kept shut. Herein arises the first difficulty between the theory and the practice of ven-

tilation, for it is plain that the simplest and often the best way to ventilate a room is through open windows. The second difficulty arises from the fact that the conditions within and without the room to be ventilated are not constant. The principal factors here concerned are the force and direction of the wind, changes of outdoor temperature, and, to a less degree, movements within the room. It is, therefore, much easier to maintain constant air conditions in a sub-basement than in a room exposed to wind and weather.

The efficiency of any system of ventilation must be measured by the results obtained at the breathing zone. It matters little what the composition or the condition of the air is near the ceiling, provided the heated, moistened, and vitiated aerial blanket which surrounds us is constantly removed and replaced with a fresh supply properly conditioned.

Ventilation is far from satisfactory if the air brought into the room is smoky, dusty, or bacteria-laden, or if it is contaminated with gases or odors from cellars or surroundings. Attention should, therefore, be given to the sources of the air, and it is always an advantage to filter it. There is a practical limit to the amount of fresh air that may profitably be forced into a room, especially warmed air in the winter time. Ventilation and heating naturally go hand in hand.

The belief is growing that it is not dangerous to rebreathe air, and the view is spreading that the hygienic value of ventilation for the purpose of maintaining a pure atmosphere in dwellings, schools, and hospitals is not so great as is commonly supposed. According to this view it is more important to ventilate in the interest of the heat economy of the body, by the establishment of a suitable temperature and air movement, and by the regulation of the humidity in the atmosphere. The established facts, that the principal causes of the ill effects of vitiated air are due more to the heat and humidity and stillness of the air than to changes in its chemical composition, have led some hygienists to recommend rebreathing the air, provided the physical conditions are kept favorable. This is an extreme view, in which I do not concur. Because rebreathed air has not been demonstrated to be harmful is no proof that it may not be so. Satisfactory ventilation, therefore, must not only take into account the physical conditions of the air, but also demands a generous supply of fresh air in order to keep the chemical composition within reasonably normal limits.

Dwelling houses are usually constructed with little regard for ventilation. It is desirable that adequate provision should be made for the ventilation of every house that is built. This requires just as much care and forethought as the system of heating the house, or furnishing it with water, gas, electricity, plumbing for the disposal of wastes, and other household conveniences. Whatever system of ventilation may be

adopted, it is wise to flush rooms frequently with fresh air and flood them with sunshine. This helps to blow out the accumulated dust and bacteria, to oxidize organic matter that collects as a film on all surfaces, to diminish odors, and generally to purify the apartment.

Vitiation by Respiration.—An adult individual at rest breathes at the rate of about seventeen respirations a minute. At each respiration about 500 c. c. (30.5 cu. in.) of air pass in and out of his lungs. The air in the lungs loses about 4 per cent. of oxygen and gains 3.5 to 4 per cent. CO_2. The nitrogen remains unchanged. In addition the expired air is raised in temperature to nearly that of the blood, 98.4° F.; it also contains much aqueous vapor.

The amount of CO_2 which is given off by an adult male person at rest can be calculated from the above figures, and will be found to be 0.68 cubic foot in one hour. Thus:—

$$\frac{17 \times 30 \times 60}{1728} = 17.2 \text{ cubic feet breathed per hour.}$$

4 per cent. of 17.2 = 0.68 cubic foot per hour of CO_2.

From actual experiment it has been determined that an average adult gives off 0.9 of a cubic foot of CO_2 during gentle exertion, and possibly as much as 1.8 during hard work. The adult female gives off about one-fifth less under similar circumstances, and an infant is said to give off about 0.5 cubic foot of CO_2 per hour. In a mixed assembly at rest, including male and female adults and children, the CO_2 given off per head is, therefore, taken as 0.6 of a cubic foot.

The volume of air inspired and expired depends on the rate and extent of the respiratory movement, but in an adult man of average size and vigor about 500 cubic centimeters of air are inspired and expired during quiet breathing. This volume of air is known as the *tidal air,* and, since the total volume of air in the lungs is about 3,500 c. c., it is evident that in normal breathing a large amount of air—3,000 c. c.—remains in the lungs at the end of expiration. The air which remains behind is known as *stationary air.*

By forced expiration about half of the stationary air, i. e., 1,500 c. c., can be expired, and this portion of the stationary air is known as the *supplemental or reserve air,* while the final 1,500 c. c. which no effort can expel is known as the *residual air.* The total of 3,500 c. c. of air in the chest, then, at the end of ordinary inspiration is made up as follows:

Tidal air		500 c. c.
Stationary air	Supplemental or reserve	1,500 c. c.
	Residual air	1,500 c. c.
		3,500 c. c.

When, however, inspiration is forced, another 1,500 c. c. of air, known as *complemental air,* can be inspired, making altogether 5,000 c. c.

The total amount of air (complemental, tidal, supplemental) which can be inspired after forced expiration is known as the "respiratory capacity" or "vital capacity" or "extreme differential capacity," and the amount varies considerably according to height, weight, vigor, age, etc.

The Amount of Air Required.—Omitting from consideration the question of temperature and moisture, a certain amount of pure air is necessary for good ventilation. This amount is determined from the amount of carbon dioxid taken as an index of the impurities from respiration and combustion, and may be ascertained either by direct observation or from physiological data. The accepted amount of pure air required per person per hour is from 2,000 to 3,000 cubic feet. The external air contains 3 parts of CO_2 per 10,000 (0.03 per cent.), and the permissible limit for indoor air is placed at from 6 to 10 parts.

It has been found from actual observation that an adult in an air-tight compartment will vitiate the air as follows:

In a	room	3,000	cubic	feet	CO_2=0.06	per	cent.	in	1	hour
"	"	2,000	"	"	"=0.07	"	"	"	"	"
"	"	1,500	"	"	"=0.08	"	"	"	"	"
"	"	1,200	"	"	"=0.09	"	"	"	"	"
"	"	1,000	"	"	"=0.10	"	"	"	"	"

The same results may be obtained from physiological data. Thus, the average adult expires 0.6 cubic foot of CO_2 per hour. The difference between the permissible limit, 0.06 per cent., and the amount of carbon dioxid in the air, 0.03 per cent., is 0.03. It follows that the amount of fresh air required per hour by an adult to keep the CO_2 down to 0.06 per cent. may be determined from the following equation:

$$0.03 : 0.6 :: 100 : x$$
$$x = 2{,}000 \text{ cubic feet.}$$

If the normal amount of carbon dioxid in the air is taken as 0.04 instead of 0.03, the result is 3,000 cubic feet, the amount generally accepted, which, however, is somewhat in excess—as it should be. This does not mean that there should be 3,000 cubic feet for each person in an inhabited room, for it is sufficient if the air-space is 1,000 cubic feet, provided, of course, the air is changed three times an hour.

The same results may be obtained by using the formula:

$$\frac{E}{P} = D$$

E=the amount of carbon dioxid exhaled by one person in one hour; the general average for an adult being 0.6 cubic foot.

P=the amount of added CO_2 permitted, stated in cubic feet; or 0.06—0.03=0.03 per cent., or 0.000,3 cubic foot.

D=the required delivery of fresh air in cubic feet per hour.

$$\frac{E}{P} = D, \text{ or } \frac{0.6}{0.0003} = 2{,}000 \text{ cubic feet.}$$

The primary value of E in this equation varies with different conditions.

A male adult	(160 pounds)	exhales 0.72 cubic foot of CO_2	per hour
A female adult	(120 pounds)	exhales 0.60 cubic foot of CO_2	per hour
A child	(80 pounds)	exhales 0.40 cubic foot of CO_2	per hour
Average		0.60	

These values vary also with rest or work. Thus, factories or workshops where men are actively employed need more air proportionately. In light work a man weighing 160 pounds exhales 0.95 cubic foot, while at hard work 1.84 cubic feet, of CO_2 per hour.

The formula suggested by DeChaumont is $D = \frac{A}{B-C}$

A=quantity of CO_2 given off per hour per person=0.6 cu. ft.

B=proposed permissible maximum quantity of CO_2 per 1,000 cu. ft.=0.6 per 1,000.

C=amount of CO_2 present in 1,000 cu. ft. of fresh air (0.3 cu. ft. per 1,000 cu. ft.).

D=amount of fresh air required per head each hour to maintain the standard B expressed in thousands of cu. ft.

$$\text{Then } D = \frac{A}{B-C} \text{ or } \frac{0.6}{0.6-0.3} = \frac{0.6}{0.3} = 2{,}000 \text{ cu. ft.}$$

of air needed per head per hour.

In case of individuals doing light work and giving off 0.95 cu. ft. CO_2 per hour, then

$$D = \frac{0.95}{0.6-0.3} = 3{,}166 \text{ cu. ft.}$$

This is a convenient formula, for it may be used not only to determine the amount of fresh air required, but, knowing the other factors, the amount of cubic feet of fresh air that has been admitted to a room

per head may be determined. Further, probable conditions of the atmosphere of a room into which a known amount of fresh air has been supplied can be determined by finding the value of B, thus:

$$(B = \frac{A}{D} + C).$$

Standards of Purity—Efficiency of Ventilation.—There is no single standard by which the purity of the air or the efficiency of ventilation can be determined. We must know at least five factors: (1) the temperature; (2) the humidity; (3) the movements of the air; (4) the amount of CO_2 it contains; (5) dust, bacteria, gases, etc. In a general way it may be stated that the best results are obtained when the temperature is between 62° and 68° F.; the moisture between 50 and 75 per cent. relative humidity (the wet bulb under 70° F.); the movement gentle, without draft; CO_2 not in excess of 6 parts per 10,000; and, finally, freedom from excessive dust, bacteria, gases, etc. Even where all these factors are found satisfactory there is still one test that must be made in order to be sure that our ventilating system is nowhere at fault—that is, the clinical test. Persons occupying the room should suffer from none of the well-known effects produced by air in poor condition. The room should be free from unpleasant odors. If our tests seem right, but the air is close, something must be wrong with the tests. The evidence of our senses and clinical experience cannot be disregarded.

Where any ventilating device is installed it is readily possible to measure, by means of the anemometer, the amount of air passing through inlets or outlets, but it is often difficult to trace the course of the air in the room. The measured volume of air passing through inlets and outlets does not necessarily determine the efficiency of ventilation in maintaining a continuous renewal of the air at the breathing zone.

The volume of fresh air entering the breathing zone may be estimated with considerable accuracy by determining the proportion of CO_2 which this zone contains. The air supplied is inversely as the respiratory contamination. It may be computed from the following equation:

$$A = \frac{vp}{x - N}$$

v=the CO_2 produced by one person; that is, 0.6 cubic foot per hour.
p=the number of people in the room.
x=the proportion of CO_2 per cu. ft. in the inside air.
N=the proportion of CO_2 per cu. ft. in the outside air (0.0003).
A=the air supplied to the room in cubic feet per hour.

$$A = \frac{0.6\ p}{x - 0.0003}$$

It will be seen in this equation that vp represents the CO_2 produced by occupants and x—N represents the respiratory contamination.

In such computations, as also in the direct measurement of air supplies, it is the averages which are most important. From average contamination we may find average air supplies. Erroneous conclusions are very likely to be drawn from single determinations.

Another method of determining the efficiency of ventilation is intentionally to vitiate the air of a room, and then, after a lapse of a certain time, find how far ventilation has removed the carbon dioxid. The amount of air which has entered the room may be found by the formula:

$$C = 2.303\ m\ \log \frac{P_1 - a}{P_2 - a}$$

C=amount of air which has entered; 2.303 is a constant.

m=capacity of the room.

P_1=amount of carbon dioxid originally present (found by experiment).

P_2=amount of carbon dioxid present after vitiation.

a=amount of carbon dioxid in the outside air.

The Size and Shape of the Room.—These are exceedingly important factors in any system of ventilation. It at once becomes evident that a man in a diving suit with a good circulation of fresh air is better off than occupants of a spacious but poorly ventilated apartment in which the air has become vitiated through long occupancy. The air in a small cabin on a steamship may be infinitely better than the air in a large room of a country home. A rathskeller in the subbasement may, with a modern system of ventilation, have much better air than that found in a department store with acres of floor space and high ceilings. In other words, a small space is sufficient if properly ventilated; a large space inadequate if improperly ventilated.

The size of rooms for dwellings and workshops is somewhat of an economic question, but they should be large enough to allow the air to be replaced two or three times an hour without causing perceptible drafts. The minimal space, in accordance with this standard, is about one-third the quantity of air required per hour; that is, from 700 to 1,000 cu. ft. per person. The amount of space naturally varies with dwellings, factories, schools, theaters, prisons, hospitals; also with the length of time the room is occupied and the nature of the work there carried on. Thus, in hospitals where ordinary cases are cared for, from

1,800 to 2,000 cu. ft. of air is desirable for each patient, while no less than 2,500 cu. ft. should be allowed for each fever patient. Soldiers in barracks are allowed 600 cu. ft. per head, and the limit for lodging houses is usually fixed at from 300 to 500 cu. ft. The U. S. Emigration Law requires 500 cu. ft. per head in the steerage. In figuring the amount of air space in a room allowance should be made for furniture, projecting surfaces, and other objects which diminish the available space. The following table from Parkes and Kennwood shows the attempts made by Great Britain to fix the minimum space allowed per head by legislation:

	Minimum Space per Head in Cu. Ft.	Authority
Common lodging houses (sleeping rooms)	300	Local Government Board (Model By-laws).
Registered lodging houses—		
Rooms occupied by day and night	400	Ditto.
Rooms occupied by night only	300	Ditto.
Non-textile wrokrooms	250	Factory Act, 1901.
Non-textile workrooms during overtime	400	Ditto.
Underground bakehouses	500	Order under Factory Act, 1901.
Above-ground bakehouses where night work is carried on by artificial light other than electric light	400 between 9 p. m. and 6 a. m.	Ditto.
Army barracks	600	British Army Regulations.
Army hospital wards	1,200	Ditto.
Public elementary schools	80	Educational Department.
London County Council Schools	130	London County Council.
Canal boats (persons over 12 years)	60	Local Government Board, Regulations under the Canal Boat Act, 1877.
Canal boats (persons under 12 years)	40	
Seamen's cabins	72	Merchant Shipping Act.
Cows in cowsheds	800	Local Government Board, Model Regulations under the Dairies, Cowsheds, and Milk-shops Order.

A little consideration, however, will show that such regulation of space is by itself of little value. Unless there be movement of air, space alone is futile. However large the space may be, the air will become impure unless fresh air circulates through it, and however small the space the air may be kept pure by sufficient circulation.

As the result of many analyses that have been made by Haldane and Osborne, they found that the carbon dioxid bears no relation to the amount of air space under practical conditions. In fact, the most

highly vitiated air found was in a room with an air space of about 10,000 cu. ft. per person.

It is not alone the air space but the shape of the room that influences ventilation. It is a mistake to suppose that a lofty room is, therefore, an airy room, for a stratum of warm vitiated air soon occupies the upper portion of such a space, and, so far as good air is concerned, has the effect of lowering the effective height of the ceiling to the top of the door or nearest outlet. Anyone may convince himself of this fact by getting up on a stepladder in a room with a high ceiling, improperly ventilated, and occupied for some hours. The upper stratum of air in such rooms is frequently stifling. Ordinarily 12 feet is high enough for the ceiling of school rooms, museums, hospitals, etc., and 9 feet for the rooms of private dwelling houses. Where there is little or no movement of the air it soon becomes offensive, no matter what the height of the ceiling.

Floor space is more important than height. The necessity for an abundant floor space is shown by the fact that a small inclosure with four high walls and without a roof, if crowded, speedily becomes oppressive. In fact, the four walls are not necessary to demonstrate this, for "crowd poisoning" in the open air upon a still, warm day is a common experience. According to Harrington, when the allowance is only 500 cubic feet per inhabitant, the floor space should be 42 square feet ($8\frac{1}{2}$x$5\frac{1}{2}$). In the English barracks the soldiers are allowed 50 square feet of floor space. For school rooms the British Educational Code requires 120 cubic feet per child in average attendance and a floor space of 10 square feet.

Inlets and Outlets.—Whether a room is to be ventilated by natural or mechanical means, proper inlets for the fresh air and outlets for the vitiated air must be provided. No general statement as to the best size and position of these openings will apply under all circumstances.

Knowing the velocity of the incoming air, the area of the inlets may be proportioned so as to permit the movement of the necessary amount of air. The size of the openings under specified conditions is, therefore, a matter of simple arithmetic. In measuring the effective area of inlet and outlet tubes allowance must be made for friction and for the guards or fretwork which protect the openings. These often diminish the effective area about one-half.

It is usually better to admit the incoming air into a large apartment through a number of openings rather than through one large one; the same holds true of outlets. Outlets should be about the same size as inlets and should be placed with reference to them.

All air ducts tend to become soiled with dust and soot and should, therefore, be guarded with wire gratings, muslin, porous flannel, or other protecting and filtering devices, and they should also be cleaned

periodically; further, it should be borne in mind that ventilating ducts are favorable highways for mice, roaches, and vermin. Inlets opening upon the floor are objectionable, as they collect unusual amounts of dirt and dust, which are then blown into the room.

Whether the air is to be admitted near the floor and taken out near the ceiling or *vice versa* is a question much discussed among ventilating engineers. Various possibilities are shown in the diagram, Fig. 89. The natural course of the warmed vitiated air is upward, and

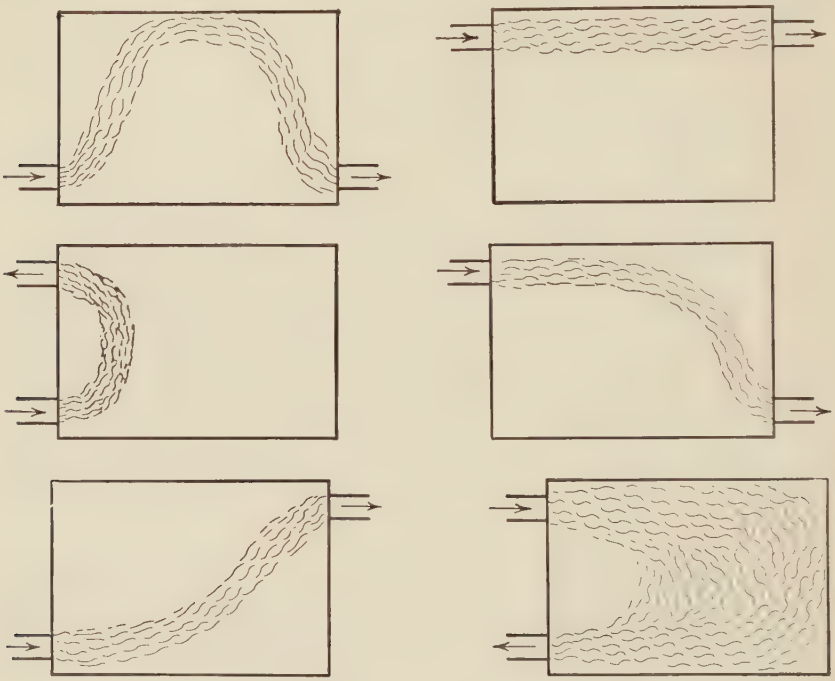


Fig. 89.—The Position of Inlets and Outlets, and Their Relation to the Air Currents in a Room.

it would seem that the upward system has advantages over the downward system. However, a little study will soon convince one that if the incoming air is warm it will rise at once, and the maximum efficiency will be lost at the breathing line, which, after all, is the essential stratum of air in the room. Perhaps the best arrangement is to have the inlet above and the outlet below—both upon the same side of an inner wall.

Outlet ventilation may be arranged by placing a bell cover or glass globe over the gas lights and conveying the heated air thence to the outer air by means of ascending tubes. This not only removes the products of combustion, but, if the outlet tubes have a sufficient area,

affords a very good system of ventilation. An automatic system of taking the air out of a room may also be provided by placing a shaft either around the chimney flue or against one side of it. The column of heated air in the ventilating duct will rise and draw the vitiated air out of the room with which it is connected. The same may be accomplished by placing a steam jet or a gas burner within the ventilating duct to create a draft.

Ventilating ducts usually extend up the walls of the building through the roof, and should be in as direct a line as practicable. The openings upon the roof may be protected by an umbrella-like covering against rain, or they may be cowled to prevent down drafts. It appears that none of the exhaust cowls cause a more rapid current of air than prevails in an open pipe under similar circumstances.

External Ventilation.—Model city planning should provide streets of sufficient width, and should regulate the height of buildings and also limit the extent upon which the land may be built, so as to allow a free circulation of air about all structures and admit a flood of sunshine for at least a few hours during the day. Some of our metropolitan streets resemble canyons rather than city thoroughfares. Crowded tenements facing upon narrow streets with shafts for courts and backing almost directly upon the houses in the rear, and further surrounded by tall buildings which prevent the free movements of the outer air, and shut out the sunshine, should be prohibited, whether used as dwellings or workshops. In such places the ground stays moist, the air becomes stagnant, natural ventilation is greatly retarded, and the conditions upon a hot, still, moist day in summer become almost intolerable.

Generous parks, which are the lungs of a great city, should be scattered throughout the residential and business sections; playgrounds, boulevards, and small open areas treated as parkings not only beautify but help to ventilate a city and add to the comfort, happiness, and health of its inhabitants.

Natural Ventilation.—Natural ventilation depends upon openings, such as doors and windows, also upon the air that comes through the pores of plaster, brick, and stone and through floors and ceilings and through the cracks and crevices about window frames, etc.

Natural ventilation depends mainly upon three principal factors: (1) perflation and aspiration; (2) gravity or thermal circulation; (3) diffusion of gases. These factors constantly operate, whether in the presence or absence of any mechanical system. In fact, most schemes for mechanical ventilation are simply an application of these natural forces.

Perflation is simply the blowing of the air into the room as a result of the movement of natural air currents. Aspiration is the sucking

action of the wind which draws air out of a space that it is blowing across. Thus, a wind blowing across an open tube carries along with it some of the air in the upper part of that tube. This causes an upward movement of the air in the tube. The same phenomenon takes place when wind blows by a window. The aspirating action of air is well demonstrated in the construction of an ordinary atomizer.

The air is kept in almost constant motion through changes in temperature. Warm air expands, is therefore lighter, and rises. This is a familiar phenomenon in the hot-air balloon. Thermal circulation, though often imperceptible, is constantly in operation, especially in occupied rooms. Even in calm weather there is considerable ventilation owing to differences in temperature, and hence differences in pressure between the air of the room and the outside.

More air than is commonly supposed enters or leaves a room through the cracks about doors and windows and other crevices. From the standpoint of natural ventilation it is, therefore, not advisable to have windows fit too snugly. The use of weather-strips, tongue and grooved metal strips, and similar devices to keep out the cold air saves coal bills, but is a considerable hindrance to natural ventilation.

Under certain conditions very considerable amounts of air pass through the building materials used in the construction of walls, floors, and ceilings. Ordinary mortar is most permeable, then comes brick, then sandstone, next plaster of paris, while enamel and tile are impervious. Under a pressure of 108 millimeters of water the following amounts of air pass in one hour through one square meter of:

Mortar	3,264	liters
Plaster of paris	146	"
Bricks	312-1,396	"
Sandstone	426- 496	"

A pressure of 108 millimeters of water is equivalent to the pressure of a strong wind. The amount of air that will pass through porous materials varies, of course, with the temperature, moisture, and other factors.

Märker and Schultze, in their researches on the spontaneous ventilation of stables, found that the following interchange of air occurred per hour over one square yard of free wall at 9.5° F. difference of temperature:

With walls of sandstone	4.7	cu. ft.
Quarried limestone	6.5	" "
Brick	7.9	" "
Tufaceous limestone	10.1	" "
Mud	14.4	" "

It is possible to force sufficient air through an ordinary brick to deflect the flame of a candle on the other side. This demonstration

is usually accomplished by coating the edges and exposed portions of the brick with sealing-wax and arranging glass funnels on either side. Air forced with a bellows through one funnel may be measured either as to its amount or velocity as it comes out of the opposed funnel.

Natural ventilation is better in winter than in summer, owing to greater differences in temperature. It may be almost *nil* on a hot calm day. Too much moisture in the air of a room settles upon the surfaces and thus stops the pores of building materials, and also prevents the escape of carbon dioxid. Rain has a similar effect on the outside. An ordinary brick will soak up a pint of water. Ventilation through the walls is also hindered by oil and enamel paints and by wall-paper. Outside obstacles, such as excessive foliage and narrow streets, are also considerable factors.

Natural ventilation may be greatly favored by simple devices. This may be demonstrated by placing a lighted candle in a bottle with a narrow neck. The flame soon dies out, but by placing a partition in the neck of the bottle, so that the products of combustion will escape on one side and the fresh air enter upon the other, natural ventilation proceeds so that the candle remains lighted. There are numerous simple devices that may be placed at the top or bottom of windows which favor the entrance of fresh air or the exit of vitiated air. An arrangement shown in Fig. 90 gives very satisfactory results. One of the upper window panes may be valved or fitted with a fan to permit the entrance of fresh air or the exit of vitiated air. Openings in ceilings, ridged ventilators, Sheringham's valves, Ellison's bricks, Tobin's tubes, and Stevens' drawer-ventilator are all useful accessory devices to aid natural ventilation.

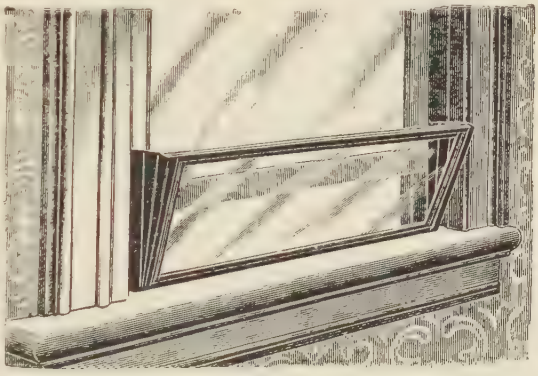


Fig. 90.—Window Ventilator.

Ellison's bricks are bricks with conical perforations, the widened end of the conical opening debouching on the interior of the wall. The holes through the bricks are about 2/10 inch in diameter externally and 1¼ inches internally.

Tobin's tube consists of a large upright tube, about 5 or 6 feet high, which conducts outside air into the room through the wall.

The Sheringham valve is a small vertical flap door in the wall near

the ceiling, balanced by a counterpoise, and hinged so as to fall forward toward the room; it is cased in at the sides and front, so that the current can only pass upward.

Stevens' drawer ventilator is like a drawer lacking its back. It is made to fit into a hole in the wall in such a way that when the drawer is shut the hole is airtight, and when the drawer is open air can enter.

Hinckes-Bird ventilator is made of the opening between two ordinary window sashes when the lower is raised, and the lower opening

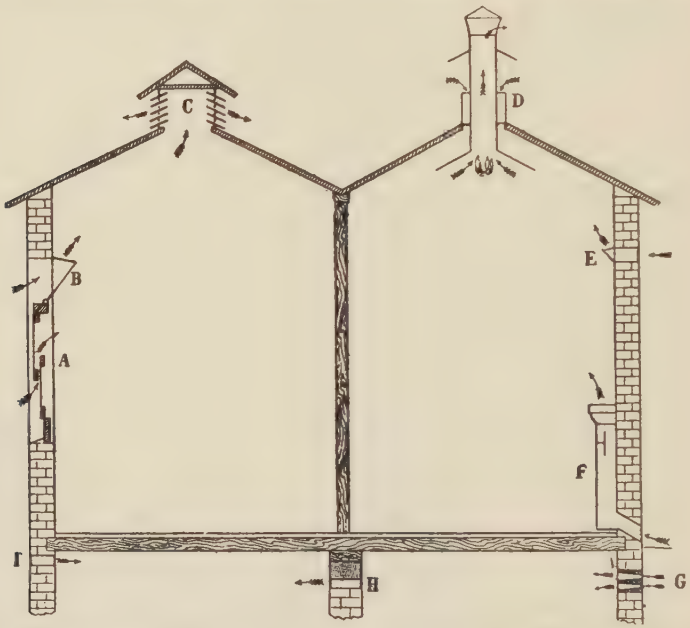


FIG. 91.—DIAGRAMMATIC SKETCH OF VARIOUS PROVISIONS FOR VENTILATION.—A, Sash window with Hinckes-Bird's arrangement. B, Hopper sash-light falling inwards. C, Louvred outlets. D, McKinnell's ventilator. E, Sheringham's valve. F, Tobin's tube (showing valve open). G, Ellison's conical bricks. H and I, Grid ventilators below floor joists. (From "Hygiene and Public Health," by Drs. L. C. Parkes and H. R. Kenwood, London, H. K. Lewis, Philadelphia, Blakiston, 1911.)

closed by means of a specially high sill or by an accurately fitting block of wood.

These various devices should be protected with valves so that they may be regulated. Sometimes it is advisable to provide gauze or cotton filters to keep out the dust.

Natural ventilation is greatly aided by means of warming the air in the outlet duct. The best example of this is the open fireplace, or other devices for warming the air in outlet tubes already referred to.

Wherever possible, open windows are the best and simplest means of ventilating a room. Any system of mechanical ventilation at best is costly and frequently unsatisfactory. Open windows are cheap and adequate, but the limitations and disadvantages of natural ventilation

are obvious, and, therefore, we are frequently required to resort to mechanical means.

Mechanical Ventilation.—All "artificial" systems of ventilation depend upon one of three methods: (1) plenum system, which consists in the mechanical propulsion of air into the room; (2) the vacuum system, which consists of the mechanical extraction of the air out of the room; (3) a combination of the plenum and vacuum systems.

Air may be propelled into a room either by means of a warming apparatus or by mechanically propelling the air by means of rotary fans. Every heating apparatus is secondarily a ventilating device, especially hot-air furnaces, and the direct-indirect systems in use with hot-water or steam pipes. Stoves, open fireplaces, and similar heating arrangements are also good ventilating devices in that, if well constructed, they take out large quantities of air from the room.

For the mechanical propulsion of air either fans or "blowers" are used. These may be run by electricity, gas, or steam power. The air is forcibly driven through ducts to where it is wanted. Without this system of mechanical ventilation the great office buildings, basement restaurants, large passenger steamships, and other modern structures would not be habitable.

If dependence is placed solely upon drawing the vitiated air out of a room we are leaving to chance where the fresh air is coming from to replace it. In other words, it is impossible when the so-called vacuum system alone is used to control the source of the fresh air and insure its purity. As a rule, all well-ventilated structures depend neither upon the plenum nor the vacuum systems alone, but combine the two.

The disadvantages of the mechanical systems of ventilation are that they are expensive as to first installation and as to maintenance; furthermore, they are designed to work only when all the doors and windows are kept closed. The advantages are that they are effective in all kinds of weather, and require less space for the air ducts than natural ventilation.

HEATING

Heating and ventilation go hand in hand. A large share of the cost of heating is chargeable to ventilation, hence, if ventilation is overdone, it is an unnecessary expense. The artificial warming of houses has a similar action to clothing. "Burning fuel in the furnace saves fuel in the human machine." It especially saves the strain upon the metabolism of the young, the old, and the feeble. The tendency in winter is to wear too much clothing indoors in order to compensate for our imperfect systems of heating. This results in coddling—that is, loss of vasomotor tone of our peripheral capillary circulation, from the constant bathing of the skin in a close moist layer of air. This in

turn results in susceptibility to drafts and liability to colds. It is quite unnecessary to wear heavy winter clothing in rooms and offices properly heated and ventilated.

Most of our American houses are overheated with abnormally dry air in the winter time. This is a mischievous combination. It causes excessive evaporation from the skin and mucous membranes, which gives rise to a feeling of chilliness. It also causes dryness of the skin and mucous membranes, irritation of the throat, and thus predisposes to colds and respiratory infections. Warm dry air does not give the same sense of warmth and comfort afforded by a cooler moist air. Thus, air at 62° to 65° F. and a relative humidity of 70 per cent. feels warmer than air at 70° to 72° F. and a relative humidity of 50 per cent. or less. Furnace heat, hot-water, and steam pipes tend to dry the air, and thus it becomes necessary to overheat our offices and houses before they become comfortable.

Heat is measured by the British thermal unit (B. T. U.), which is the quantity of heat required to raise the temperature of a pound of pure water one degree at its point of maximum density, 39° F. The French thermal unit is the calorie and is the amount of heat required to raise one kilogram of water one degree centigrade at corresponding temperature (4° C.). One calorie equals 3.968 B. T. U.

Heat travels by radiation, conduction, and convection. All three routes are constantly in operation in any system of heating. Thus, with an open fireplace the heat waves radiate in straight lines to the nearest objects, where they are absorbed or reflected, just as light passes through space independent of the atmosphere. That is why our face toasts and our back freezes before an open fireplace. The heat absorbed by any object passes through that object from particle to particle by conduction. Most metals are good conductors; air is a very poor conductor of heat. Convection is the process by which heat is communicated through gases and liquids as a result of their mobility. Thus, the air is warmed by our bodies, by hot-water pipes, and by all heated objects, and therefore rises and establishes convection currents.

There are five main methods of heating: (1) open fires; (2) stoves; (3) hot air; (4) hot-water or steam pipes; (5) electricity.

Open Fires.—The open fireplace heats mainly through direct radiation. It has the advantage of being cheerful and a good ventilator. It has the disadvantage of being wasteful and very unequal if depended upon as the chief source of heat.

Parkes and Kenwood estimate that, in an ordinary medium-sized sitting room with an ordinary fire, from 10,000 to 15,000 cu. ft. of air are drawn up the chimney in an hour, the current being generally from 3 to 6 ft. a second. "As ventilating agents," say Notter and Firth, "the best types of open fireplace cause some 2,600 cu. ft. of air

to pass up the flue per pound of coal consumed, or the passage of about 18,000 cu. ft. up the chimney per hour."

Franklin Stoves.—Franklin stoves consist of coal fires in a cast-iron stove, the products of combustion being carried off through a stove-pipe. Such stoves, standing free in the room, are very efficient, so far as heating is concerned, and also favor ventilation through the circulation of air, which is drawn into the stove to support the burning of the fuel. The heating of the room is unequal, as it depends largely upon radiation and somewhat upon convection. Such stoves are apt to become red-hot, in which case it is believed they allow carbon monoxid to pass through the cast iron. The organic dust in the air falling upon the hot stove is burned and produces an unpleasant smell.

Open Gas Heaters.—Open gas heaters without flues to carry off the products of combustion are bad, from a sanitary standpoint. These heaters consist of a series of metal tubes containing air or water, which are heated with naked flames. The heat is thus imparted to the room by convection and also by radiation. Such devices may contaminate the air with carbon monoxid from leakage or from unconsumed gas, or from the formation of soot, which becomes incandescent. Such heaters also contaminate the room with CO_2 and other products of combustion. The "rubber" tube feeding these gas heaters often leaks, and there is frequently a perceptible odor of gas in rooms where these devices are used. Open heaters burning oil are less objectionable than those using gas.

Hot-air Furnaces.—A hot-air furnace consists of a coal fire which heats a series of tubes or plates in the dome of the furnace. The air, which is usually taken from the outside through a duct, flows into this dome, where it comes in contact with very hot surfaces, and is thus conducted by thermal circulation through a series of ducts into the rooms of the house. A hot-air furnace of this kind constantly pumps fresh air into the house and is, therefore, a very efficient system of ventilation. The objection to the hot-air furnace is that the air is excessively dry and frequently partly "burned" in passing over the heated surfaces in the dome. The odor caused by the burning of the organic particles in the air may frequently be noticed in houses heated with a hot-air furnace. The heated air entering the rooms is usually allowed to escape as it will. In order to overcome the disadvantage of the dryness of the air furnished by the hot-air furnace, water pans are always provided, from which the water is supposed to evaporate. These pans are ridiculously small and cannot possibly furnish sufficient moisture for the great volume of air constantly passing through one of these furnaces. For instance, according to Harrington, air at 25° F. saturated with moisture and then heated to 70° F. would need half a pint in every thousand cubic feet to give it a humidity of 65 per cent. The

air from a hot-air furnace is perhaps drier than that furnished by any other system of heating or ventilation. Thus, an out-of-door air in winter at a temperature of 0° F., with a relative humidity of 50 per cent., when heated to 70° F., will have a relative humidity of only 3 per cent. This is drier than the air of the driest climate known, which is seldom less than 30 per cent. It is not unusual for the excessively dry air of a furnace-heated house to cause the woodwork to shrink and fall apart, the bindings of books to crack, etc. Living in such an atmosphere is not normal and must be harmful.

Hot-water and Steam Pipes.—This is a very simple and effective system of heating buildings. The hot-water system is especially applicable to small buildings and steam pipes to large buildings. The hot water is more readily controllable than steam, which has a tendency to overheat. Special furnaces are found on the market to heat the water or to generate the steam, which then circulates through pipes to the rooms where wanted. If the hot-water radiators or steam coils are exposed directly in the room, the system is known as the "direct." In the direct-indirect system the hot-water pipes or steam coils are placed in a special box where the air from the outside is heated, and this heated air flows by thermal circulation through ducts into the rooms where wanted. In the direct system the air of the room is simply heated and reheated over again, while in the direct-indirect system the fresh warmed air is constantly pumped into the building and it is, therefore, an efficient method of ventilation. In both these systems the air is abnormally dried, just as it is in the hot-air furnace, though not to the same degree.

Electric Heating.—Electric heating is clean, easily regulated, but expensive. It has the disadvantage of being insufficient as a ventilating device, unless special inlets and outlets are provided. Electric heaters consist simply of resistance coils which heat the room mainly through radiation and convection.

The Cooling of Rooms.—Much attention has been given, through necessity, to the heating of rooms in winter time, but heretofore little attention has been given to the cooling of rooms in the hot season. It is quite as practicable to cool rooms as it is to heat them, and sometimes quite as important to health.

The principle of practically all cooling devices depends upon the fact that when a fluid evaporates to its gaseous state it absorbs a considerable amount of heat—latent heat. This heat is taken from the surrounding objects which, therefore, become correspondingly cold. Cold may also be produced by the expansion of air. This was pointed out in 1845 by Joulé. Thus, if a jet of air at 60° F. were blown into a room under a pressure of 10 inches of mercury above the ordinary barometric pressure, the sudden expansion of this compressed air would

reduce it to a theoretical temperature of 13.3° F. below freezing. This principle of dynamic cooling has been applied to refrigerators.

Ammonia gas is now almost universally employed in freezing machines. This gas is readily condensed into a liquid. The compressed gas is allowed to expand into tubes, and the cold thus produced utilized directly; more frequently an indirect method is used by which the expanding gas first cools a freezing mixture consisting of a saturated solution of calcium chlorid; this chilled brine is then pumped through a series of pipes to the refrigerator or apartment where it is desired.

A simple method of cooling a room is by the rapid evaporation of water. Dr. Manning was able satisfactorily to cool a large room in the Government Printing Office at Washington by blowing air by means of an electric fan over a moist sheet. This sheet, about a yard wide, was hung near the ceiling, and constantly wetted by a stream of water flowing over it.[1]

[1] Many of the facts in this chapter upon "Ventilation and Heating" are taken from Donald C. Macfie's book on "Air and Health," 1909, published by E. P. Dutton & Co.

SECTION V

SOIL

CHAPTER I

GENERAL CONSIDERATIONS

The upper layer of the earth's crust, known as the soil, is derived from the disintegration of rocks and the decay of animal and vegetable matter of all kinds. It varies from a few inches in depth to several feet. The sub-soil also varies from a few feet to hundreds of feet in depth, to hard pan or an impermeable stratum.

From a sanitary standpoint the soil must be regarded as our friend rather than our enemy. Enormous quantities of organic matter and infections of all kinds find their final resting place in the soil and are there disposed of and rendered harmless by nature's beneficent processes. In fact, a closer study of the functions of the superficial layer of the soil shows that it is not only the organ of digestion and respiration of the earth, but, like the liver, it is the great organ in which toxic substances of all kinds are neutralized or destroyed.

The sanitarian does not look upon the soil as dead and inert, but rather as a living being, for it presents many of the vital phenomena that characterize life: digestion, metabolism, assimilation, growth, respiration, motion, and even reproduction. The soil breathes, it absorbs oxygen and exhales carbon dioxid; it is capable of digesting and assimilating vast amounts of organic matter by a complex process of metabolism; the waste products are excreted. If these wastes are retained the soil may be choked or killed by an accumulation of its own poison—a sort of autointoxication. The soil, like all living things, demands water, but it may be drowned by an excess. A water-logged soil dies in very much the same sense that an individual dies who has suppression of urine. Sedgwick speaks of the "living earth" in the sense that it is teeming with life; bacteria, molds, amebæ, and many of the primitive forms of the animal kingdom, as well as worms, insects, snakes, birds, rodents, and many other animals, make their temporary or permanent homes in the upper layers of the earth. Earth worms by their

plowing action, so beautifully shown by Darwin in 1881, constantly turn over the upper layers of the earth. The soil, therefore, is in constant peristalsis, which helps its digestive functions. The rise and fall of the ground water is analogous to the movements of the diaphragm and assists the respiratory functions of the soil.

Classification of Soils.—Soils are variously classified, depending upon the amount of sand, gravel, clay, loam, humus, peat, muck, rock, alkali, etc., which they contain. The difference between a sandy and gravelly soil depends mainly upon the size of the particles. These soils interest the sanitarians because hookworms live and flourish in them better than they do upon clay or rock formations. "Clay exists in particles of the smallest possible size. It is very cohesive, possesses a high degree of plasticity, and plays a very important part in determining the fertility of soils, their texture, and their capacity for holding water. Its plasticity is due to the presence of a small proportion of hydrated silicate, and is modified very greatly by the addition of less than a hundredth part of caustic lime. It is exceedingly impermeable to water, and when wet dries with great slowness" (Harrington). Loam consists of a mixture of sand, clay, and humus. If the sand predominates the soil is said to be light; if the clay predominates, heavy. A rich soil contains an abundance of humus.

By humus is meant the products of vegetable decomposition in their various intermediate stages of decay. It is the essential element of vegetable mold, and is necessarily of most complex composition. It is composed of a great number of closely related definite chemical compounds, chief among which are ulmin and ulmic acid, which are supposed to characterize brown humus; humin and humic acid, which dominate dark or black humus; and crenic and apocrenic acids. Humus contains a high percentage of nitrogen, especially marked in some of our prairie soils and in the "black soil" found in the provinces of the Ural Mountains, which, according to von Hensen, contains as much as from 5 to 12 per cent. of organic matter.

Surface Configuration.—Geodesy, or surface configuration, has an important relation to health. Low and swampy ground is a breeding place for the malarial mosquito. Highlands are apt to be drier and more healthful than lowlands. A slope affords better drainage than flat lands, and thus diminishes the dangers from soil pollution, but increases the risk of infection being washed down from those living above. In narrow valleys the air stagnates, the moisture is excessive in both the soil and the air, and there is an unpleasant blanket of cold layers of air at night. Mountain sides are notoriously windy. High plateaux suffer from extremes of temperature. Thus, at Mexico City (about 8,000 feet above sea level) there is a sharp contrast between the temperature during the day and night, and even during the daytime be-

tween the sunshine and the shade. At Quito, which is 9,350 feet above the sea level, the daily variation of temperature at some periods of the year is no less than 34° F. Northern exposures do not get enough sunshine, and southern exposures sometimes too much.

The relation of the surface configuration of the land to health is intimately interwoven with the whole question of climate, and must take into consideration temperature, air movements, humidity, sunshine, barometric pressure, precipitation, and the seasons with their endless varieties from tropical to arctic.

Composition of the Soil.—Much attention was formerly given to the hygienic importance of the chemical constituents of the soil. The presence of organic substances was regarded not only with suspicion, but as a serious menace to health. It was claimed that organic pollution of the soil made a good culture medium for the germs of infectious diseases. The gaseous products of decomposing organic matter in the soil have long been looked upon as particularly injurious. These gases, with other ill-defined but unknown volatile substances, are spoken of as miasma or effluvia.

We now know that very few, if any, of the bacteria pathogenic for man grow and multiply in the soil under natural conditions. The spores of tetanus, malignant edema, and anthrax may live in garden earth for many years, but it is doubtful whether these microorganisms, especially the anaerobes, ever find conditions favorable for growth and multiplication in the soil. Ordinarily typhoid, dysentery, and cholera bacilli do not flourish in the soil; on the contrary, they soon die there. It has been shown that cities built upon polluted soils have sometimes suffered relatively less from typhoid and cholera than cities built upon rocky or virgin soil. In some cities (as Budapest) it has been pointed out that the greatest morbidity and mortality rate was in that part of the city built upon made ground filled in with trash and much organic waste. These instances have been largely coincidences, for, as a rule, the low-lying, polluted soil happened to be the poor, crowded tenement district. A sanitarian does not recommend polluted soils for building sites, but it seems that their influence upon health has been overstated, especially where cellars are properly constructed. While a polluted soil may not be hazardous in the ways just indicated, it may be dangerous so far as hookworms and other parasites are concerned, or indirectly it may lead to contamination of drinking water, food, etc. See "Pollution of the Soil," page 682.

Mineral Matters in the Soil.—By far the most abundant element in the soil is oxygen. According to various estimates, from 33 to 50 per cent. of the solid crust of the earth consists of oxygen. The other elements found in abundance in the soil are: silicon, carbon, sulphur, hydrogen, chlorin, phosphorus, fluorin, aluminium, calcium, magnesium,

potassium, sodium, iron, manganese, and barium. Aluminium silicate or clay makes up perhaps two-thirds of the inorganic components of soils. Other compounds are lime and magnesia carbonates (limestone) and numerous chlorids, sulphates, phosphates, oxids, etc., of the various bases.

Iron is universally present and gives the red color to soils. Nitrogen exists in soils in three distinct forms: (1) protein and its split products, (2) ammonia and its salts, and (3) nitric acid and nitrates or nitrous acids and nitrites.

Vegetable Matter in the Soil.—The vegetable matter exists in the soil in various stages of decomposition. One result of the decay of vegetable substances is the formation of well-defined compounds, such as ulmic, humic, and apocrenic acids. These organic acids have considerable power to dissolve mineral substances, which accounts in part for the plumbosolvent action of acid-reacting surface waters from swampy lands.

Peat or muck results from the incomplete decay of vegetable matter under water.

Animal Matter in the Soil.—Organic matter of animal origin in soils results chiefly from the decomposition of carcasses or from contamination with the excreta of animals. As a rule, animal matter is neither so abundant nor so widely distributed in the soil as vegetable matter. From a sanitary standpoint soils polluted with organic matter of animal origin present a greater danger than soils polluted with vegetable matter.

Physical Properties.—In general it may be said that the physical properties of a soil are more important, from the standpoint of health, than its chemical composition.

Porosity.—By the porosity, or pore volume, of a soil is meant the volume of the interstices between the particles, which may be filled with water or air, or both. Ordinarily the pore volume in soil amounts to about 40 per cent.; some apparently compact masses, such as sandstone, have as much as 30 per cent. The pore volume of the soil is independent of the size of the individual grains.

Permeability.—The permeability of a soil to air does not depend upon the pore volume, but upon the size of the individual pores.

Water Capacity.—The water capacity of the soil is the amount of water held in the interstices of the soil when saturated, while the *water-retaining capacity* is the amount of water held back after a saturated soil is drained.

Soil Temperature.—The sun is the principal source of the soil temperature. Some heat is produced from chemical changes, but not in considerable amounts. The original heat of the earth's interior furnishes a constant source of heat that is of much importance.

The heat absorbed and given off by the soil has a notable influence upon the atmospheric temperature. Some soils and moist surfaces absorb heat from the sun and give it off again when the sun has set. The most heat-absorbent soils are sandy soils. The sand of the desert may be heated to 200° F., and when this hot sand is raised by simoons the temperature of the air in the shade may reach 125° F. or more. The power of absorbing or reflecting solar heat also depends upon the color of the soil.

Absorption.—The soil has, to a remarkable extent, the property of absorbing odors and gases, and ordinarily it is very hygroscopic. The soil is also capable of holding toxins, colors, and other substances through the physico-chemical property of absorption. In this respect it acts like charcoal. Illuminating gas from leaky mains may be divested of its odorous constituents in its passage through the soil, so that its presence in houses may be undetected, thereby greatly increasing the danger. In the experiments made by Abba, Orlandi, and Rondelli about the filtering galleries of the Turin water supply the property of the soil to hold back substances in solution was shown. Cultures of *Bacillus prodigiosus* in large volumes of water poured into the ground at various points made their appearance 200 meters away in 42 hours, whereas dyes, such as methyleosin and uranin, could not be detected until after 75 hours.

Soil Air.—Air is present in all soils, even in the hardest rocks. Sandstone may contain from 20 to 40 per cent., sand from 40 to 50 per cent., and humus as much as 2 to 10 times its own bulk. The soil air differs markedly in composition from that of the atmosphere. It is usually very moist and contains various gases, especially carbon dioxid, resulting from the decomposition of organic matter. For the same reason soil air contains less oxygen than the free atmosphere. The soil air varies greatly, according to the character of the soil, the climate, the season, and rainfall. There is a continual interchange between the air of the soil and the air of the atmosphere. This interchange is influenced by differences in temperature, by rainfall, and by the movements of the ground water and by barometric pressure. Rain chokes the pores and checks soil ventilation. The soil air is in constant motion.

Following the teachings of Pettenkofer, the amount of carbon dioxid in the soil air was for years taken as an index of the amount of soil pollution. It is now well known, however, that this is not a reliable index, for the reason that many conditions influence the amount of CO_2 in soil air. A soil recently manured may contain from 2 to 5 or even 10 parts of CO_2 per thousand. In a gravelly soil the proportion may be as high as 80 parts per thousand.

Soil air may influence health when contaminated with poisonous

gases, such as carbon monoxid. This occasionally happens. In the open these gases would be so greatly diluted that they could scarcely exert a deleterious influence, but when concentrated, as they sometimes are in dwellings, and breathed for a long period of time they may be responsible for anemia, headache, and other symptoms. Soil air containing carbon monoxid may be sucked into a dwelling from long distances in a lateral direction. Leaky gas pipes may thus render the air of a dwelling impure if the cellar is permeable. This is favored by the pumping action of the furnace, especially when the surface of the ground is frozen.

Soil air is practically sterile; that is, under ordinary conditions it contains few bacteria. Odors sometimes contained in the air from a polluted soil have no known injurious effect.

Soil Water.—The passage of water through the soil is essential to soil activity. The moisture favors the bacterial growth by which soils purify themselves and favors vegetation. Nitrates, chlorids, and other soluble substances are dissolved in the water and pass into the subsoil, or furnish food to the roots of plants. A soil absolutely dry, as a desert soil, is lifeless. A soil with an excess of moisture, that is, one in which the ground water level is at or near the surface, delays and alters the natural decomposition of organic matter. In the deeper layers of the soil, where no bacterial action takes place, vegetable matter may remain almost permanently without change. Thus, wooden piles are not attacked after centuries.

Water exists in the soil in two principal forms: (1) *soil moisture,* which comprises the water present in the interstices of the upper partly saturated layer, as well as the watery vapor contained in the soil air, and (2) *ground water,* or sub-soil water, in which case the interstices of the soil are completely filled.

The soil moisture is estimated by determining the loss of weight by drying 10 grams of soil at 100° C. to constant weight. The dry sample may then be exposed to air saturated with moisture under a bell jar and again weighed. The increase in weight indicates the absorptive power of the soil.

Water may also be regarded as existing in the soil under three conditions, viz., hygroscopic, capillary, and gravitation. The hygroscopic water is that which adheres to the surface of the soil particles in the presence of air. The capillary moisture is that which is held within the spaces that are capillary in their nature. The gravitation water is that which drains through the soil and accumulates in the subsoil over an impermeable stratum. For a discussion of ground water see chapter on water.

It is generally stated that a persistently low ground water level, viz., 15 to 20 feet, is healthful, and that a persistently high ground

water level, viz., 3 to 5 feet, is unhealthful, and that a ground water level that fluctuates suddenly is still more unhealthful. Pettenkofer found that typhoid fever was more likely to occur at Munich, Berlin, and Leipzig when the ground water level was at its lowest. His explanations to account for this were ingenious, but we now know that the relation was only a coincidence, for the same does not hold in other places.

Sub-soil drainage is usually considered more of an agricultural necessity than a public health question. Large tracts of our land in the middle West and in other parts of the world have normally a high ground water level, and it is necessary to bring this down in order to increase the fertility of the soil. This is done by draining the subsoil, which also abolishes marshy and swampy lands, and thus puts a check upon malaria.

One of the principal influences of the soil upon general health is through soil moisture. Dampness in or near the surface of the soil may affect the health of those dwelling nearby. Such a soil is cold, and the atmosphere immediately above it is liable to be damp, and this appears to conduce to rheumatism, neuralgia, and diseases of the respiratory tract. Investigations seem to indicate that the general health of those dwelling on damp soils is inferior to that of those more favorably circumstanced in that regard.

The Nitrogen Cycle.—The most interesting of the vital phenomena taking place in the soil is the disposal and utilization of organic matter. This may best be illustrated by the nitrogen cycle, which must be understood in order to have a clear conception of soil pollution, water purification, and sewage disposal.

The nitrogen cycle is a complex series of events which protein matter undergoes, in which it is reduced to simple and stable inorganic compounds, and then returns through plant life to the animal kingdom. One phase of the cycle, namely, the breaking down of animal and vegetable matter, is due almost entirely to bacterial action. The other phase, namely, the building up of complex living organic matter from simpler compounds and elements, is mainly a function of living plants.

The nitrogen cycle is a process in which the anabolism or synthesis occurs in plants, while the catabolism or analysis is brought about chiefly through bacterial action. Hence the series of events constituting the nitrogen cycle largely depends upon the plant kingdom. The important phases of the cycle occur upon the soil and in its superficial layer. It will presently be seen that this cycle is of fundamental importance in sanitary science, and has a special significance in preventing soil pollution, in the purification of water, and in the disposal of sewage. It is evident that any permanent break in this cycle would result in the cessation of life upon the earth.

As soon as an animal or plant dies its protein constituents are at once attacked by putrefactive bacteria. The proteolytic microorganisms growing in and upon the nitrogenous matter break it up into secondary and simpler products, which have a striking resemblance to the cleavage products of gastric and pancreatic digestion. Some of the putrefactive bacteria, of which the *Bacillus subtilis* and the *Bacillus proteus* are important types, liquefy protein matter during the process of putrefaction. Other bacteria, of which the colon bacillus is a type, break down organic matter without evident liquefaction. Very many other species of bacteria take part in this stage of the cycle. For the most

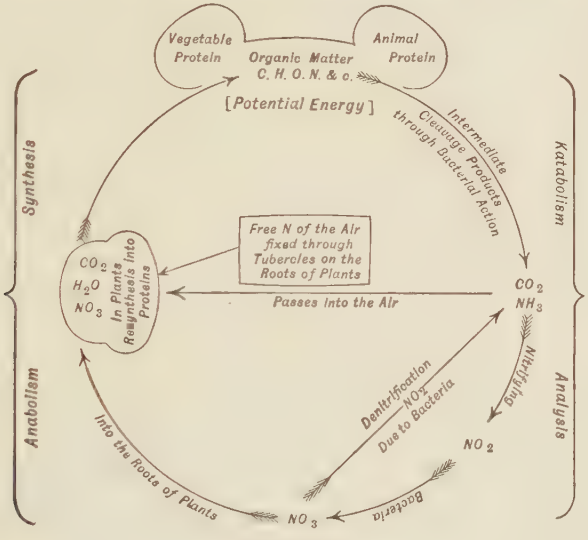


Fig. 92.—The Nitrogen Cycle.

part the microorganisms pathogenic for man are killed during the process of putrefaction; they die in the struggle for existence. The processes of decomposition are essentially the same, whether the organic matter is the carcass of an elephant, a beetle, a tree, or a leaf, provided that the necessary moisture, warmth, and other conditions for bacterial growth are present. The breaking down of vegetable matter is slower and more difficult than the breakdown of animal matter. This is due in part to the fact that the latter contains larger percentages of putrescible protein and also usually contains more moisture, which favors bacterial activity.

The breaking down of the complex protein molecules to simpler and stabler compounds is usually spoken of as mineralization, and may be regarded as a series of oxidations. According to our present chemical conception, it is really a series of hydrolyses. The complicated

molecular structure of protein matter is analyzed into amino compounds of simpler and simpler composition, until nitrogen finally appears in the form of ammonia. We know little of the chemistry of the early stages of protein decomposition. The process seems hopelessly complicated from the intricate structure of the molecule. Eventually from the seething caldron of molecular disintegration there appear simpler substances, such as proteoses, peptone, ptomains, amins, leucin, and tyrosin, and other amino substances, as well as organic acids, indol, skatol, phenol, and finally sulphuretted hydrogen, mercaptan, carbonic acid, and ammonia. One of the final products of the process is carbon dioxid, part of which passes into the atmosphere and part of which is retained in the soil as carbonates of alkalies or alkaline bases. The

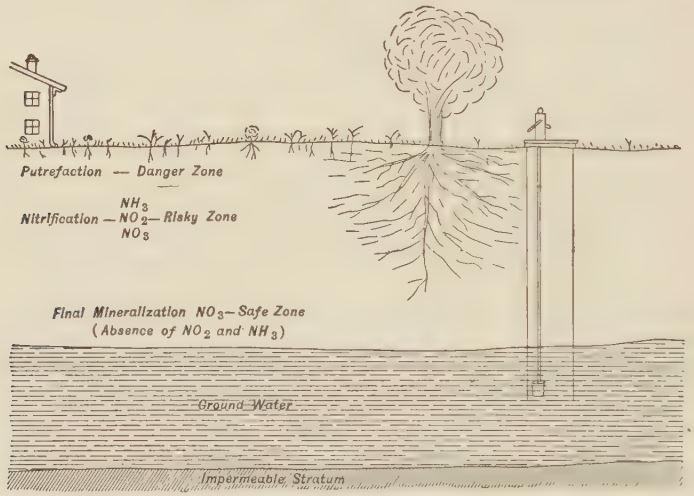

Putrefaction — Danger Zone
Nitrification — NH_3 NO_2 NO_3 — Risky Zone
Final Mineralization NO_3 — Safe Zone
(Absence of NO_2 and NH_3)
Ground Water
Impermeable Stratum

FIG. 93.—THE NITROGEN CYCLE IN DIAGRAMMATIC VERTICAL SECTION.

ammonia, as such, cannot be used by plants. Some of it may escape into the atmosphere, but for the most part it is retained in the soil as ammonium chlorid or ammonium carbonate. In the soil the ammonia is oxidized by the action of nitrifying bacteria into nitrates. This nitrifying action of bacteria, elucidated by Winogradski in 1888, was one of the brilliant discoveries in bacteriology. Through his work and that of later workers, it is now known that this process is usually accomplished in two distinct steps. In the first stage the ammonia is oxidized to nitrous acid. This is done by the nitrosobacteria. These nitrous or nitrite bacteria were called by Winogradski nitrosomonas and nitrosococcus. It is now known that a large number of microorganisms belong to this group. The nitrites exist in the soil probably as salts of potassium and sodium. They remain as the lower oxid a very short time and, therefore, never accumulate, and are never found in any large

amount for they are unstable and readily oxidized to nitrates. The special nitric or nitrate bacteria (nitrobacter) were first accurately described by Winogradski. The nitrates are stable and represent the final stage of the mineralization of nitrogenous matter. In certain arid parts of the world large deposits of nitrates (KNO_3, saltpeter) are found as the result of the nitrification of bird excrement (guano), which is rich in available nitrogen. These collections do not occur in places where there is enough rain to carry away the readily soluble nitrates.

Ordinarily the nitrates go into solution in the soil moisture and are either taken up by the roots of plants or are washed away in the ground water. In a sanitary analysis of water taken from the soil the presence of nitrates and nitrites, therefore, has a special significance. If nitrites are found in soil water it indicates pollution and signifies active bacterial action and the presence of organic matter. Nitrates in soil water, without nitrites, are an index of past pollution (see Water Analysis).

In 1886 Gayon and Dupetit described two organisms, *B. denitrificans* α and β, capable of completely reducing nitrates. Many bacteria have this power of denitrification, a sort of reversible process by which nitrates are reduced to ammonia. This is characteristic of very many of the well-known microorganisms, such as the colon group, pyocyaneus, subtilis, and other soil bacteria. Denitrification, however, does not occur to any notable extent in a well-ventilated soil.

In plant metabolism the nitrates are used to build up new protein. Certain plants get some of their nitrogen through the bacterial tubercles on their roots, which have the power of fixing the free nitrogen of the air. These small nodules are abundant on the roots of various leguminous plants (peas, clover, etc.). Pure cultures of the legume or nitrogen fixing bacteria, such as *Bacillus radicicola* of Beyerinck, may be obtained from these root tubercles.

It should be noted also that certain bacteria (azobacter) have the ability to fix the free nitrogen of the air independently of plant life and may grow under either aerobic or anaerobic conditions. One of the first known of this group was an anaerobe described by Winogradski in 1895 and named by him *Clostridium pasteurianus.*

It will be noted that in the nitrogen cycle all the essential steps from proteolysis to mineralization of the organic matter, nitrification, oxidation, and reduction, as well as the fixation of free nitrogen from the atmosphere, are all the result of bacterial action. Each stage of the complex process is specific, in the sense that it requires a particular species or group of bacteria to affect the result, and also specific in the sense that special conditions of environment are necessary for its action to take place.

It is important to remember that practically the entire cycle takes place upon the surface and in the upper layers of the soil. A few

feet below the surface of an undisturbed area the soil contains few or no bacteria. Carcasses buried deep, or sewage placed too far below the surface, do not profit by the nitrogen cycle in its entirety, and under such circumstances incomplete nitrification takes place. Nature's method of disposing of dead wastes is thereby defeated, and pollution of the soil and infection of the ground water may result.

The Carbon Cycle.—Carbohydrates, such as cellulose, starch, sugars, and similar constituents of vegetable and animal matter, are fermented, with the formation of carbon dioxid, alcohol, and various organic acids. The carbon in carbohydrates passes through a series of changes, which may be regarded as the carbon cycle. The carbon dioxid resulting from fermentation unites with water in the plant life, and under the action of chlorophyll and sunlight is again synthetized to starch and sugars.

The fermentation of the carbohydrates is also due to the action of microorganisms. In a mixture containing both carbohydrates and protein, as a rule, the bacteria act upon the carbohydrates first. In other words, the putrefaction of protein is delayed or hindered by the presence of fermentable carbohydrates. For this reason the disposal of sewage containing wastes from breweries is difficult.

Fats are also attacked by bacteria, with the consequent production of acids. The hydrocarbons are broken down with more difficulty than either the carbohydrates or protein. An excessive amount of fat in sewage always gives trouble on a filter. For instance, the drainage from a wool-scouring mill containing lanolin and the discharges from slaughter houses and the wastes from creameries and cheese factories containing animal fat present special problems in sewage disposal.

CHAPTER II

THE SOIL AND ITS RELATION TO DISEASE

Bacteria in Soil.—Countless millions of bacteria occur in the upper few inches of the soil. The enormous overgrowth of bacteria in the upper layers of the soil gives it the sticky, moist feeling which rich soils possess. The odor of the soil, such as that which is particularly noticed after a rainstorm, is due in large part to *Cladothrix odorifera* and other organisms which are commonly found in the soil. Few bacteria are found in an undisturbed soil below a depth of 4 to 6 feet. A sand bed used for filtering sewage shows a similar vertical distribution of bacteria. Below six feet the statement is made that the soil is usually sterile. This is not strictly true, but the numbers are much diminished and bacterial activity has practically ceased. As a rule, living bacteria are not obtained from samples of soil obtained 10 to 12 feet below the surface, except in soils with large pores or crevices, or in cases where the bacteria have been carried by burrowing animals. It is exceedingly difficult to determine the number of bacteria in the soil, as so many of them are anaerobes and vast hordes belong to the nitrifying groups, which grow only upon selective media. The soil is also the home of other species, requiring special conditions for growth in artificial culture media.

Of the ordinary bacteria that grow upon the usual laboratory media Houston found an average of 100,000 per gram in an uncultivated sandy soil, 1,500,000 per gram in a garden soil, and 115,000,000 per gram in a sewage soil. Peaty soils have smaller numbers. The actual numbers must be vastly greater, for many microorganisms in the soil do not grow upon the common media. In fact, the soil is the home of the greatest number and variety of bacteria found anywhere. It is the bacteria in the upper layers of the soil that make it resemble a living gland. Each particle of earth is coated with a zoogleal envelope. The sand and mineral particles form the supporting structures, the coating of bacteria corresponds to the glandular epithelium, and the interspaces between the particles are the capillary and lymph channels.

Most of the bacteria in the soil are saprophytes. The microorganisms pathogenic for man do not find conditions favorable for growth and development in the soil. For the most part the tempera-

ture is too low; further, they are crowded out by the overgrowth of the saprophytes. Koch has demonstrated that anthrax and other pathogenic bacteria may be grown in sterile soil, but cannot be grown in unsterilized soil, that is, in living soil. They die in the struggle for existence. Experiments have shown that the soil of graveyards contains no more bacteria than the corresponding soil in the same locality, and is noticeable by the absence of pathogenic microorganisms. The soil often contains the bacteria (or their spores) of certain wound infections, such as malignant edema, anthrax, *B. aerogenes capsulatus,* and tetanus. The relation of the soil to typhoid, cholera, dysentery, hookworm disease, Cochin-China diarrhea, and other infections will be discussed presently.

The function of the bacteria in the soil may best be understood by studying the fate of organic matter polluting the soil and the processes which accomplish its purification (see Nitrogen Cycle, page 676).

Pollution of the Soil.—The soil is capable of disposing of great quantities of organic matter. However, if it is overburdened it remains polluted and may endanger health through contamination of the drinking water and in other ways. It is not only the amount but the kind of pollution, and also the manner of its disposal, that plays a very important part. It must first of all be remembered that the purifying action of the soil is largely dependent upon bacteria, and that this action takes place almost solely in the upper layers. If carcasses are buried deeply, or if sewage is allowed to enter the soil at several or more feet below the surface, the process of purification is long delayed or checked. A leaky cesspool or broken drain which discharges its contents into the soil at a depth of 5 feet or more may seriously pollute the ground water, whereas the same material placed upon or just beneath the surface may be entirely mineralized and all infection destroyed before it reaches the depth of 5 feet. Vegetable matter in a water-logged soil undergoes a partial and unusual decomposition into muck or peat. Trees buried deeply, where bacterial action is practically absent, remain for many hundreds of years practically unchanged. Many factors retard the purifying action of the soil. Among these the temperature and moisture and absence of oxygen predominate.

When organic matter falls upon the soil it is consumed and digested by the hungry earth. Without this property the surface of the earth would long ago have become clogged with vegetable and animal matter. Albuminous substances are dissolved by the action of the proteolytic bacteria, and converted into simpler chemical compounds. The intermediate products of protein putrefaction are exceedingly complex. For our present purposes it is sufficient to know that ultimately the nitrogen is largely converted into ammonia and the carbon into carbon dioxid. The ammonia is then oxidized by the action of nitrifying

bacteria to nitrites, and the nitrites again oxidized to nitrates. The nitrates are the final products of the mineralization of organic matter. Most of the nitrates pass into solution and are carried down into the deeper layers of the soil or sub-soil; some of it is taken up through the roots of plants. The carbon dioxid passes off into the air as a gas, remains in the soil moisture in solution, or is converted into carbonates.

Pathogenic bacteria that may be thrown upon the soil in feces or otherwise are usually detained in the upper layers and finally destroyed there. Under ordinary conditions pathogenic microorganisms are caught in the upper layers of the soil, just as they are caught upon the "schmutzdecke" of a slow sand filter. The soil does not act simply as a mechanical trap. The bacteria are detained and destroyed by a combination of physical, chemical, and vital processes taking place in the upper layers of the soil.

All polluted soils are not equally dangerous. Soils polluted with human feces and urine present the greatest hazard to man. The special menace of soils polluted with human excreta is from typhoid bacilli, hookworms, and other infections discharged in the feces or urine. Hookworm infection is usually contracted directly from soils polluted with human feces, and the eradication of hookworm disease depends primarily upon preventing pollution of the soil. The danger in the case of typhoid, dysentery, cholera, and other bacterial infections is usually indirect through infection of drinking water or occasionally through flies or other mechanical means of transference. A soil polluted with typhoid may endanger either the surface water or the ground water, particularly in limestone formations. Pathogenic microorganisms in a polluted soil may also find their way back to man upon vegetables. Tapeworms and other intestinal parasites pass part of their life cycle on or in the soil, and may infect man directly or indirectly in various ways. The question of soil pollution and the particular ways in which it is related to health have been discussed separately under each disease concerned.

Dirt.—The soil is often spoken of as dirt. The soil in the field is "earth," but in the parlor or on our hands it becomes dirt; that is, matter out of place. The word "dirt" is from the old Saxon "drit," meaning excrement. Dirt in the ordinary sense becomes a potential danger, especially when containing human excretions or soil bacteria associated with wound infections.

To the sanitarian dirt includes rubbish, manure, and organic wastes of all kinds. It may be the vehicle, but not the source, of infection. It breeds and harbors flies, fleas, lice, rats, mice, and vermin of all sorts that act as intermediate hosts or carriers of infection. While dirt cannot originate typhoid fever or other infections, it favors conditions which encourage the spread of such diseases. Rubbish in vacant lots,

in backyards, in alleys, in cellars, garrets, and other places may be taken as an index of the failure to appreciate the modern teachings of hygiene and sanitation. It was once the chief duty, and still an important one, of the health officer to insist upon cleanliness of premises and surroundings, both in country and city.

Cleanliness.—Cleanliness is the heart and soul of sanitation. We are inclined to place it even before godliness, for cleanliness of body, cleanliness of mind and soul, and cleanliness of our surroundings are essential to a full appreciation of the spiritual virtues. Our conception of cleanliness has greatly changed with our advance in knowledge of the kinds of dirt, the degrees of dirtiness, and the nature of these dangers. We can no longer be satisfied with physical or esthetic cleanliness, but must insist upon biological cleanliness. A tetanus spore upon the shining blade of a surgeon's knife makes that instrument filthy, whereas many such spores on the skin of a chicken may be harmless. We cannot see the infection upon the common drinking cup, upon the roller towel, upon the point of a pencil that has just been moistened with saliva, or in water, milk, or food, although we well know the danger of such invisible "dirt" that these objects may harbor.

It requires a bacteriologist to tell the difference between clean dirt and dirty dirt. We lack a sixth sense, or microscopic eye, to see and distinguish the harmful germs. We, therefore, must practice scrupulous cleanliness and educate the people to the biological meaning of this term. Long experience has taught the lesson that cleanliness offers a protection against disease; that clean surroundings are apt to be free of infection; and that clean food is apt to be safe food.

At one time the theory of the filth diseases reached the dignity of a special name—the pythogenic theory, first propounded by Murchinson in 1858. Typhoid fever was long regarded as the type of a filth disease, and, while we are now dropping that term, we should not forget that typhoid fever is really a filth disease, for each case means that a short circuit has been established between the discharges from one person and the mouth of another.

The Influence of the Soil upon Health.—The soil was formerly accused of being one of the largest and most important factors in the spread of the communicable diseases. It was once regarded as the cause, if not the nesting place, of infections of all kinds; tuberculosis, malaria, typhoid fever, plague, yellow fever, cholera, dysentery, and many other diseases were directly associated with the soil. We now know that comparatively few of the microorganisms pathogenic for man live in the soil, and practically none of them grow and multiply there.

The soil contains a number of bacteria that may be serious when

introduced into wounds, as tetanus, malignant edema, anthrax, *B. aerogenes capsulatus;* ofttimes organisms belonging to the hemorrhagic septicemic group; sometimes staphylococci and streptococci.

A soil polluted with human excrement presents the possibility of danger of intestinal infections of all kinds. Thus, bacterial infections, such as typhoid, cholera, and dysentery, or protozoal infections, such as amebic dysentery, or the higher worms, such as hookworms, may all more or less be associated with polluted soils.

Soils containing much organic matter and presenting other favorable conditions afford resting and nesting places for a number of insects, such as flies, ticks, etc., which may carry infections.

Vegetables grown in polluted soils may transfer bacteria, protozoa, or the eggs of worms in a mechanical way from the ground to the mouth. This applies particularly to vegetables eaten raw, such as radishes, lettuce, etc.

Practically all the water used for drinking and other purposes has either rested upon the soil or has percolated through it into the ground. The soil materially affects the character of the water. In this way the soil indirectly influences health variously and sometimes seriously. The relation of water to health is a subject in itself, and is discussed in a separate chapter.

The physical conditions of the soil which have special reference to health are those which influence the temperature and moisture of human habitations. Persons working about cold and damp soils are subject to rheumatic, neuralgic, and respiratory affections.

Diseases Associated with the Soil.—Tetanus.—Spores of the tetanus bacillus commonly occur in the soil of inhabited regions. They have been found not only in the superficial layers, but sometimes at a depth of several feet. The normal habitat and the great reservoir of tetanus are the intestines of the herbivora. It may also be found in the intestinal contents of man and other animals. Certain savages in the New Hebrides used to smear their arrow heads with dirt from crab holes in the swamp, which they knew by experience to be poisonous. We now know that this material contained tetanus spores.

Tetanus increases as we approach the tropics, where puerperal tetanus and tetanus of the newborn are relatively frequent. Tetanus spores are much more abundant in certain localities than others. For example, certain parts of Long Island and New Jersey have become notable for the number of cases of tetanus caused by small wounds.

The tetanus bacillus probably does not grow and multiply in the soil. It cannot there find the necessary anaerobic conditions, temperature, and other factors necessary for multiplication. The resistance of the spores accounts for the persistence of the infection.

The prevention of tetanus has been discussed on page 66.

Anthrax.—Like tetanus, anthrax does not grow in the soil under natural conditions. Its persistence is accounted for by its resistant endospore. Anthrax spores have been found in pastures where infected animals have been confined.

The anthrax bacillus requires oxygen in order to sporulate; the spores, therefore, do not form in the blood, and it is very important not to open the carcass of a sheep or cow dead of this disease before it is buried. The classic researches of Pasteur on anthrax should be studied in this connection. Pasteur examined the field where animals dead of anthrax had been buried twelve years previously. He found the specific bacillus in the soil and demonstrated its virulence by inoculations into guinea pigs. Pasteur thought that the spores were brought to the surface of the soil by earthworms, and proved the possibility of this by sowing virulent cultures in soil and recovering the bacillus from worm casts. It seems, however, in the light of subsequent investigations that the danger from this source is negligible, so that anthrax, with a few exceptions, can hardly be called a soil infection. This is the case at least with man, for there is no instance on record in which human anthrax has been contracted from contact with the soil.

Malignant Edema.—The bacillus of malignant edema is found in the superficial layers of the soil. It is very widely distributed. This organism is also found in putrefying substances, in foul water, and in the intestinal tract of various animals. In 1877 Pasteur first recognized an organism belonging to this group by injecting animals with putrefying liquids. He called the organism the *vibrione septique*, recognized its anaerobic nature, but did not obtain it in pure culture. Koch and Gaffky in 1881 studied it carefully and renamed it the bacillus of malignant edema. The bacillus has lateral flagella, an oval spore, and is a strict anaerobe. It is very pathogenic for almost all animals, causing extensive hemorrhagic edema without the production of gas, which distinguishes it from the gas bacillus of Welch. Wound infections with malignant edema occur, especially with deep punctured or lacerated wounds, which favor anaerobic growth. Before the days of antisepsis this complication was frequent, especially during wars.

Welch's gas bacillus (*B. aerogenes capsulatus*) has a similar relation to the soil to that just described. Many other microorganisms, especially those belonging to the hemorrhagic septicemic group, occur in the soil and occasionally complicate wounds.

Typhoid Fever.—There is a widespread belief, even among sanitarians, that this disease is frequently connected with soil pollution. This belief was given scientific confirmation by Pettenkofer, who propounded the theory that the poison, whatever it may be, is introduced

into the soil where, under proper conditions of organic filth, temperature, moisture, etc., a special fermentation takes place. Pettenkofer believed that the gases or effluvia thus produced rise, and in some way were capable of provoking disease. Pettenkofer's views of typhoid in relation to the height of the ground water have already been mentioned.

Typhoid bacilli frequently find their way upon and into the soil along with human excreta. Multiplication, however, rarely takes place there. As a rule, the typhoid bacillus scarcely lives a month, possibly two or three months, in the soil. When frozen they may live and remain virulent for several months, as in the case of the Plymouth episode and the New Haven epidemic. While typhoid fever in cities and towns has no evident direct relation to soil pollution, it is possible to conceive an indirect relation in many cases, especially in camps and in rural districts.

There are numerous ways by which typhoid bacilli may be returned from the soil to the mouth of a susceptible person. It is possible, though not likely, for this to occur directly. So far as typhoid is concerned, perhaps the greatest danger from a polluted soil consists in infection of the drinking water. The ways in which this may occur are discussed in the chapter on water. The transfer of typhoid bacilli from the soil to the mouth may also occur mechanically by means of flies, dust, and dirt. Vegetables grown in a polluted soil may carry typhoid bacilli to the very tips of their leaves.

The pollution of soil with human feces is always a danger and should be prevented. The worst offense in this particular occurs in country districts, where the potential danger is greater than in the city.

Cholera.—There is every reason to believe that the cholera vibrio dies quickly when deposited upon or in the soil under natural conditions. The cholera vibrio may be transferred from the soil to the mouth in the ways mentioned above in the case of typhoid. Formerly cholera was believed to be associated with polluted soils, but it now appears that the disease is rarely contracted from the soil, and that the physical and chemical conditions of the ground play little, if any, rôle in the epidemiology of this disease.

Tuberculosis and Other Diseases.—In 1862 Dr. H. I. Bowditch formulated the law of soil moisture from studies which seemed to indicate that tuberculosis was more common in Massachusetts over moist soils than dry ones. If there is any connection between tuberculosis and the soil, the relation must be indirect. Exposure to cold and damp depresses vitality and lowers resistance to tuberculosis. It does not necessarily follow that habitations or workshops are cold and damp because the ground on which these houses are built is wet and cold.

The soil was formerly accused of being responsible for plague,

malaria, yellow fever, and a long list of other diseases. The importance of the soil with reference to the communicable diseases diminishes with our increase in knowledge. The number of infections directly associated with the ground are very few, and the indirect influences are less than formerly supposed. Apart from the one real danger, viz., soil pollution with human excrement, the sanitarian is now inclined to belittle the influence of the soil upon health.

Dampness and cold may favor rheumatic and neuralgic conditions, and also predispose to respiratory infections. In this way association with a cold, damp soil may be prejudicial to health. Clay soils are apt to be damp; sand and gravel soils are readily drained and may be kept dry by means of simple devices. Such soils, therefore, make the best building sites for habitations. As a rule, the foundation of a house should be at least two or three feet above the level of the ground water.

The soil greatly influences the character of the water which rests upon it and which passes through it. This will be discussed in the section on water.

Hookworm Disease.—Hookworm disease is closely associated with the soil. It may fairly be considered an infection the result of soil pollution. It occurs especially in moist sandy soils rather than on clay or rocky soils. This is due to the fact that hookworm eggs, when deposited in fecal matter, soon dry up and die upon hard rocky or clay surfaces, whereas they find favorable conditions for development upon moist sand or loam. Under these conditions the larvæ develop as far as the second ecdysis, which have the power of penetrating the skin (see page 114).

Other Animal Parasites.—In a somewhat similar sense many of the animal parasites of man are deposited on the soil and reinfect man during one of the stages of their cycle of development. Most of the intestinal parasites of man are deposited on the soil, and, after a varying journey, sometimes through an intermediate host, again find lodgment in man. In the case of trichina, for instance, man pollutes the soil with feces containing the eggs. Hogs devour this infection and return the disease to man. In a somewhat similar way the tapeworms of cattle and also some ameba and flagellates pass part of their life history upon the soil.

The *Ascaris lumbricoides* and the *Trichiuris trichiura,* two very common worms inhabiting the intestinal tract of man, have thick-shelled eggs and must rest in the soil about a month before they are infective. It requires about a month for the embryo to develop. If fresh eggs of these two worms are ingested, they pass through the intestinal tract without hatching.

Dr. Stiles has prepared the following list of animal parasites which

have some relation to the soil during some part of their life history, and may, therefore, be more or less associated with soil pollution:

LIST OF ANIMAL PARASITES OF MAN WHICH MAY BE SPREAD BY SOIL POLLUTION

I Direct (i. e., without Intermediate Host)	II Indirect (i. e., with Intermediate Host)	III Further Study on Life Cycle Desired, Probably Belongs in I or II as Marked
PROTOZOA:	PROTOZOA:	PROTOZOA:
Entamœba coli		Entamœba gingivalis sp. inq.[1] I
Entamœba histolytica		Entamœba dentalis sp. inq. I
Entamœba buccalis		Entamœba pulmonala sp. inq. I
Entamœba tetragena		Entamœba urogenitalis sp. inq. I
Paramœba hominis		Entamœba kartulisi sp. inq. I
Leydenia gemmipara (= Chlamydophrys stercorea or C. enchelys)		Entamœba miurai sp. inq. I
Trichomonas vaginalis		
Trichomonas intestinalis (=? vaginalis)		Trichomonas pulmonalis sp. inq. I
		Cercomonas hominis sp. inq. I
		Monas pyophila sp. inq. I
Eimeria cuniculi		Monas lens sp. inq. I
Eimeria hominis		Bodo urinarius sp. inq. I
Isospora bigemina		
Balantidium coli		Balantidium minutum I
		Nyctotherus faba I
		Nyctotherus africanus I
		Nyctotherus giganteus I
		Rhinosporideum kinealyi I
TREMATODA (Flukes):	TREMATODA:	TREMATODA:
	Fasciola hepatica	Gastrodiscus hominis II
Schistosoma japonicum	Opisthorchis felineus	Watsonius watsoni II
		Fasciola gigantica II
		Fasciolopsis buskii II
		Fasciolopsis rathouisi II
		Paragonimus westermanii II
		Opisthorchis noverca II
		Metorchis truncatus II
		Cladorchis sinensis II
		Cladorchis endemicus II
		Fascioletta ilocana II
		Schistosoma hæmatobium I
		Schistosoma mansoni sp. inq. I
		Heterophyes heterophyes I
		Dicrocœlium lanceolatum I

[1] "Sp. inq." = require further study.

LIST OF ANIMAL PARASITES OF MAN.—*Continued.*

I Direct (i. e., without Intermediate Host)	II Indirect (i. e., with Intermediate Host)	III Further Study on Life Cycle Desired, Probably Belongs in I or II as Marked
CESTODA (Tapeworms):	CESTODA:	CESTODA:
	Tænia solium	Hymenolepis nana I
	Tænia saginata	Tænia africana II
		Tænia confusa II
	Dipylidium caninum	Davainea madagascariensis II
	Hymenolepis diminuta	Davainea asiatica II
	Hymenolepis lanceolata	Dibothriocephalus cordatus II
	Dibothriocephalus latus	Diplogonoporus grandis II
NEMATODA (Roundworms):	NEMATODA:	NEMATODA:
Ascaris lumbricoides		Ascaris texana sp. inq. I
		Ascaris maritima II?
Toxocara canis		Trichostrongylus instabilis I
Oxyuris vermicularis		
Necator americanus		Trichostrongylus brobolurus I
		Trichostrongylus vitrinus II
Agchylostoma (or Ancylostoma) duodenale		Metastrongylus apri I
Hæmonchus contortus		Dioctophyme renalis II
		Œsophagostomum brumpti I
Trichiuris trichiura		Ternidens diminutus I
Strongyloides stercoralis		Physaloptera caucasica I
ACANTHOCEPHALI:	ACANTHOCEPHALI:	ACANTHOCEPHALI:
	Gigantorhynchus gigas (or hirudinaceus)	
	G. moniliformis	

SECTION VI

WATER

CHAPTER I

GENERAL CONSIDERATIONS

"The greatest influence on health is exerted by those things which we most freely and frequently require for our existence, and this is especially true of water and air" (Aristotle).

While water is not technically classed as a food, it is an essential article of diet. In nature water comes in contact with many surfaces and substances and, therefore, is particularly liable to contain impurities, especially as it is the most universal solvent known. Water is also a frequent medium for the transmission of infection.

From the remotest antiquity the highest value has been placed upon an abundant and pure water supply. Centers of population sprang up in ancient times around those points where it was most readily available, and great expenditures of labor and treasure were made to carry it to places where it was not naturally plentiful.[1]

Water is a prime necessity of life—not only as an article of diet, but also for the proper cleanliness of person, clothing, and things.

It is interesting to note that the number of towns in this country before 1800 having a public water supply was only 16, supplying about 2.8 per cent. of the existing population at that time. In 1850 there were only 83 public water works, supplying about 10.6 per cent. of the census population. In 1897 the total number was 3,196, supplying about 41.6 per cent. of the population. Since then the number has greatly increased, but exact information is not available.[2]

[1] The date of construction of the Appian aqueduct carrying water to Rome is placed at 312 B. C. Eighteen other aqueducts were constructed at various times until 226 A. D. The one commenced by Emperor Caius and completed by Claudius, according to Pliny, cost 350,000,000 sesterces, or about $12,700,000.

[2] Baker, M. N.: "Manual of American Water Works," 1891 and 1897.

COMPOSITION

At the close of the eighteenth century water was regarded as an elementary substance. In 1781 Cavendish discovered that, when an electric spark is passed through a mixture of 2 parts of hydrogen to 1 part of oxygen, these gases combine to form water. Since then water has been made synthetically, and separated analytically into its component constituents by various methods.

The composition of pure water (H_2O) is:

	By Volume.	By Weight.
Oxygen	1 part	8 parts
Hydrogen	2 parts	1 part

Pure water is a chemical curiosity; it does not exist in nature. All water in nature contains impurities, in solution and in suspension. Some of these impurities are organic and some are inorganic. They consist of various gases, fluids, and solid substances. The more important impurities and their sanitary significance will be considered in detail under the chemical analysis of water.

CLASSIFICATION OF WATER

From a sanitary standpoint water is either good or bad. Commonly waters are classified as pure or impure. It is not possible, however, in the present state of our knowledge, to draw a sharp line of distinction. In the classical reports of the Massachusetts State Board of Health waters are spoken of as normal or polluted. A normal water is free from direct or indirect pollution by waste products from human life or industries. The difficulty with this classification is that normal waters may differ widely in color, taste, odor, and composition, and may, therefore, be unfit for household or manufacturing purposes.

A practical classification of waters is as follows: (1) good, (2) polluted, (3) infected. A good water may be defined as one of good sanitary quality, as determined by physical inspection, bacteriological and chemical analyses, a sanitary survey of the watershed, and, finally, by clinical experience. A polluted water is one containing organic waste of either animal or vegetable origin. A polluted [1] water is a suspicious water. An infected water contains the specific microorganisms of human diseases.[2]

In Europe waters are frequently classified as potable or non-potable.

[1] Sometimes spoken of as contaminated water.
[2] Chemical poisons such as lead are not included in this classification.

Many cities on the Continent have a double water supply with faucets plainly labeled "potable" or "non-potable," the first being suitable for drinking and cooking purposes and personal use, while the second is intended for miscellaneous household and industrial uses.

According to location, waters are considered under three classes, viz., rain water, surface water, or ground water.

PROPERTIES OF WATER

Water is a clear, transparent, tasteless, and odorless fluid; colorless in small quantities; pale blue through a deep column. It freezes at 0° C. and boils at 100° C. under a barometric pressure of 760 mm. It is practically incompressible; has its greatest density at 4° C.; is a remarkable solvent. The latent heat of water and other properties that have a sanitary bearing will be considered in the succeeding pages.

Practically all substances yield to water; it is the most universal solvent known. It dissolves gases; in fact, one of the most important constituents of all natural waters is carbonic acid. Carbon dioxid is always present in the air, and all rain waters contain some of it. Still more is taken up by the water as it percolates through ground covered with vegetation. The presence of this gas increases the solvent powers of the water, enabling it especially to dissolve limestone and many other inorganic substances.

THE USES OF WATER IN THE BODY

As a rule, water is not considered a food, for it may be said to have little or no value when estimated as a force producer within the body. Much of the water which is either drunk or ingested as a part of other foods passes through the body unchanged, but some of it is undoubtedly altered or split up into elements which unite with other compounds. The nature of these processes is obscure, and as yet very little understood. Water is entitled to rank as a food because it enters into the structural composition of all foods as well as all the tissues of the body; it is an essential element of diet, even though it cannot of itself build tissue, repair waste, or produce heat or energy.

Water composes about 70 per cent. of the entire body weight, and its importance to the system, therefore, cannot be overrated. The elasticity or pliability of muscles, cartilages, tendons, and even bones is in a great part due to the water which these tissues contain. "The cells of the body are aquatic in their habits." The amount of water required by a healthy man in 24 hours is, on the average, between 1,800 and 2,100 c. c., beside about 600 c. c. taken in as an ingredient of solid foods, thus making a total of 2,400-2,700 c. c. Twenty-eight

per cent. of the loss of water from the body takes place through the skin, 20 per cent. through the lungs, 50 per cent. through the kidneys, and 2 per cent. through other secretions and the feces.

The use of water in the body may be summarized as follows: It enters into chemical composition of the tissues; it forms the chief ingredient of all the fluids of the body and maintains their proper degree of dilution, and thus favors metabolism; by moistening various surfaces of the body, such as mucous and serous membranes, it prevents friction; it furnishes in the blood and lymph a fluid medium by which food may be taken to remote parts of the body and the waste material removed, thus promoting rapid tissue changes; it serves as a distributor of body heat; it regulates the body temperature by the physical process of absorption and evaporation.

One of the most universal dietetic faults is neglect to take enough water into the system.

THE AMOUNT OF WATER USED AND WASTED

From a sanitary standpoint our aim should be to encourage a generous use of water, but to discourage waste. The conservation of pure water and the economic value of a purified water are pressing problems that a growing and expanding country must meet and solve as a matter of self-interest if not of self-preservation.

It is possible to get along with a surprisingly small amount of water. Thresh found that in a number of country places the amount used in cottages could not have greatly exceeded one gallon per person per day. This is not sufficient for modern requirements of cleanliness and health. On the other hand, where the supply is abundant and easy of access large quantities of water are heedlessly wasted.

The average amount of water per capita required for domestic purposes is usually stated at about 17 gallons a day. Rankine considers 10 gallons sufficient. Parkes found that the average amount used by a man in the middle class, who may be taken as a fair type of a cleanly man belonging to a fairly clean household, is 12 gallons per day. This includes the amount used in cooking, drinking, ablution, utensil and house washing, and laundry. Davies' estimate of 17 gallons a day is divided as follows:

Drinking, 3 pints; cooking, 5 pints	1 gal.
Ablution (including sponge bath, 2½ gals.)	5 "
Washing (laundry, 3; house, etc., 3)	6 "
Water closets	5 "
	17 "

The actual per capita daily consumption of water in some cities is, in fact, not much above this figure. Thus, Manchester uses 20 gallons

and Berlin 22 gallons a day for each individual. Some small English towns, as Saffron Walden (population 6,108), use 11 gallons per capita per day, and Melrose (population 1,300) uses 13 gallons. As a contrast to these low figures most cities in America are furnished with an extravagant quantity—Pittsburgh, 250 gallons per capita daily, Buffalo, 223, Philadelphia, 227, Washington, 218. The small amount of water used by some European cities is not an ideal to strive for under American conditions. The European figures are steadily increasing, even where all water is sold by meter. In towns having a metered supply the per capita consumption varies from 6.6 gallons daily for the lowest class of dwellings to 59 gallons for the highest class of dwellings. The following tables from Hazen's "Clean Water and How to Get It" give the per capita consumption in some American cities, contrasted with similar figures abroad.

THE QUANTITIES OF WATER SUPPLIED IN A NUMBER OF AMERICAN CITIES

Place	Year	Gallons per capita Daily	Percentage of Services Metered
Pittsburgh	1905	250	1
Buffalo	1900	233	2
Philadelphia	1905	227	..
Washington	1906	218	3
Chicago	1900	190	3
Detroit	1905	190	29
Boston	1905	151	6
Cleveland	1905	137	68
New York	1902	129	35
Newark	1900	94	21
Milwaukee	1905	91	94
Minneapolis	1904	82	42
Worcester	1900	70	94
Providence	1905	68	86
St. Paul	1900	67	28
Hartford	1906	63	100
Lowell	1905	52	69
Fall River	1905	37	97

THE QUANTITIES OF WATER SUPPLIED IN A FEW FOREIGN CITIES

Place	Year	U. S. Gallons per Capita Daily	Place	Year	U. S. Gallons per Capita Daily
London	1906	39	Berlin	1905	22
Liverpool	1902	38	Hamburg	1905	44
Paris	1901	65	Dresden	1905	26
Amsterdam	1905	37	Copenhagen	1905	27
Melbourne	1905	63	Brisbane	1906	58
Sydney	1905	39			

The amount of water[1] expressed by the per capita consumption of a community is very misleading for purposes of comparison. The figures are usually obtained by dividing the total theoretical amount of water pumped, by the population. The result, therefore, does not take into account many factors, for the actual amount of water pumped does not equal the theoretical possibilities; corrections for slip and other factors should be made. The figures also do not take into account the amount of water lost through broken pipes, leaky joints, etc. It is estimated that in some places almost half the water pumped is wasted in this way. Further, there are great discrepancies when contrasting different cities in the amount of water used for business purposes. The amount of water used in trades and manufactures varies enormously. Certain industries, such as mining, tanneries, coal washing, paper mills, breweries, wool scouring, etc., require great quantities. It is estimated that in the iron, coal, and steel regions of Pennsylvania a quantity of water representing the entire flow of the Allegany River passes through the large steel, iron, and other mills along its banks several times before it reaches the city of Pittsburgh. Therefore, unless the per capita consumption is based upon the amount of water actually measured by meter for domestic purposes, the figures of one city cannot be properly compared with those of another.

Few persons realize the immense amount of water that is wasted in almost every town. Taking it right through, probably one-half of the water supply of American cities is wasted. While some of this is unavoidable, the greater part of it could be stopped. There are three principal causes of this waste: (1) leakage from faulty mains and service pipes; (2) waste from defective house fittings; (3) waste resulting from an unmetered or unmeasured service. The first cause includes leaks from faulty mains and service pipes and all other hidden defects where the water escapes unperceived into drains and sewers or into the sub-soil. It is possible to check a large part of this waste by the use of instruments known as detectors. With these instruments leaks may be located. The detectors are of two sorts: (1) aquaphones, instruments resembling a large stethoscope, by which a trained ear may locate murmurs; (2) pitometers, instruments which measure the rate of flow in branch lines during the small hours of the night, when practically no water is used. In this way leaks, defective taps, and open stopcocks may be discovered. It requires but a moment's calculation to figure out the great number of gallons wasted by forgetting to close a stopcock. In

[1] It has been calculated that altogether the supply of Rome was 332,306,624 gallons daily, which would have been over 332 gallons per capita upon a basis of a population of one million. This calculation, however, has been based upon data furnished by Prony in 1817. Mr. Clemens Herschel has lately shown a much more probable figure for the daily water consumption of Rome, namely: 32,000,000 U. S. gallons.

some cities, such as Washington, in the winter time the water in many houses is allowed to run continuously from the cold water faucet, in order to prevent freezing.[1] The waste from this cause is enormous, and may be corrected by properly placing the service pipes so as to avoid all danger from bursting through freezing. It has been the universal experience that much water is thoughtlessly wasted where the service is not metered. The only objection to a metered service is the prejudice common to all innovations, but the advantages are soon realized and the saving is very considerable. The introduction of meters in the city of Washington during the past few years has resulted in checking the waste by reducing the total amount of water consumed one-third, making a saving of from 20 to 30 million gallons of water a day without annoyance or inconvenience to any one. This great saving did not all result from the metering alone, but was aided by the use of detectors and an efficient system of inspection, which checked waste from other causes. In Milwaukee, before meters were generally adopted, the water used per tap was 1,781 gallons per day. After the majority of houses were furnished with meters, the amount used per tap was only 644 gallons. Another notable instance of checking waste was furnished by Liverpool, where the average amount supplied daily per head was 33.5 gallons. Deacons' water waste detectors were introduced, and these, together with efficient inspection, reduced the supply to 23 gallons without any restrictions being placed upon the consumers. At Shoreditch, in England (population 87,000), the introduction of waste detectors effected in the course of three years a diminution of waste and undue consumption amounting to 720,000,000 gallons per annum. At Exeter the introduction of waste detectors reduced the waste from 75 to 12 gallons per head per day.

It is estimated by engineers that 45 per cent. of the water supplied to Manhattan and the Bronx is wasted, and that if this waste were checked the new aqueduct from the Catskills, which is costing $140,000,000, would not be needed. While it is necessary to allow a liberal supply, there is no sanitary advantage in an excess. Good clean water in large quantities is difficult to obtain and expensive. Economy and avoidance of waste are, therefore, essential.

DOUBLE WATER SUPPLIES

The question of a double supply of water, one cheap for general purposes and the other high class for personal use, has often engaged the attention of engineers and sanitarians. Ancient Rome had a sort of double supply, and Paris and other European cities have it at pres-

[1] In cities where this practice prevails, more water is used in the winter time than in the summer months.

ent. The advantages and disadvantages of the double system are evident. Even where the community served is intelligent and careful, the danger of a double system is very great, and it will probably never be resorted to except through stress of circumstances.

Sedgwick has recently suggested that cities may be given a double water supply provided the one for general use is disinfected or denatured in such a way as to discourage its use for drinking purposes. Thus, if the second supply had added to it a large amount of bleaching powder, perhaps sufficient to make it distasteful, it would at least be harmless so far as infections are concerned. The proposition is attractive, and would serve well for street washing, fire, and other purposes.

SOURCES OF WATER

We may begin the circle by considering that all water comes to us from the aqueous vapor condensed in the form of rain or snow. Of this a certain amount returns to the atmosphere by evaporation; the rest collects upon the surface of the earth or soaks into the ground. Some of it flows off in the direction of surface slope to join the ponds, lakes, rivers, or seas, or some of it may penetrate the earth to variable depths. The sources of our water supply may, therefore, be classified as: (1) rain or snow water, (2) surface water, including ponds, lakes, streams, and rivers, and (3) ground water, including springs and wells. This classification is evidently an arbitrary one, used for convenience. There is no sharp line of demarcation between rain, surface, and ground water. Rain water soon becomes surface water, and surface water quickly passes into the ground; the ground water frequently reappears as springs to form streams and lakes and other surface supplies.

Rain water is nominally the purest and may be free from all traces of organic matter, but is liable to irregularity of composition, and in built-up sections it is very difficult to collect it so as to be free from contamination and fit for drinking. Surface water from inhabited watersheds is, in its raw condition, never entirely safe for drinking purposes. Ground water obtained from the sub-soil of a catchment area, free from sources of pollution, is usually of a satisfactory character. Artesian water, which is ground water obtained from the deeper underlying strata, is often so rich in mineral matters that it is unsatisfactory for most uses. The various sources of pollution, its character, and dangers will be considered in subsequent pages.

RAIN WATER

Rain water is really "distilled water," that is, it is water that has been vaporized and then condensed. The process of distillation is one of the best known methods for purifying liquids of all

kinds. All the non-volatile substances are left behind; theoretically, therefore, rain water should approach nearer to absolute purity than any other kind of natural water. However, it receives impurities from the moment it condenses, for each droplet of mist is formed about a particle of dust in the air. The rain drop further absorbs gases, and as it drops through the air collects a large amount of the "dirt" floating in the lower portions of the atmosphere. It is a common observation how a shower will wash the air so that it becomes beautifully clear and clean. The impurities collected by the rain before it reaches the surface of the earth, while considerable in amount, are practically negligible from a sanitary standpoint. After rain touches the earth's surface it becomes, to all intents and purposes, a surface water, unless collected with special precautions to avoid contamination. If collected from a clean, impervious surface in the open country, it is the purest of natural waters. The use of rain water for drinking purposes has met with little favor by sanitarians, despite its exceptional purity, because it is so frequently collected and stored in such a careless manner that it is subject to impurities. It is true that rain water is not likely to be infected with sewage, nevertheless some of the filthiest waters used for domestic purposes come from rain-water tanks. Even casual inspection will often show that rain water collected and stored in the usual way is very far from being pure, though rarely infected.

Because rain water is soft it recommends itself for use in the laundry, and the absence of lime salts renders it desirable for cooking. On the whole, however, it is not considered as practicable as a good ground or surface water for general domestic supply.

The use of rain water stored in cisterns is the principal factor in keeping yellow fever alive in endemic foci. The yellow fever mosquito (*Stegomyia calopus*) breeds by preference in artificial containers holding rain water. It was the abolition of such breeding places that has protected Philadelphia, Boston, and many other seaports that formerly fostered the stegomyia and suffered from yellow fever epidemics (see page 212).

Usually it is advisable to filter rain water collected from the roofs of buildings, especially if situated in towns, near dusty roads, etc.

Underground filters for rain water, in order to purify it before it enters the storage tanks, are frequently provided. These filters are for the most part unsatisfactory. Either the material is so coarse that little purification is effected, or so fine that it speedily becomes clogged and useless. They rarely receive proper attention and, therefore, are apt to become filthy.

Amount.—The average annual rainfall on the globe is computed to be 33 inches. The mean annual rainfall for different portions of the United States has been tabulated by the United States Weather Bureau

to average some 30 inches. In New England and the middle states it amounts to 40 inches. In Assam from 600 to 805 inches have been recorded, while in the Sahara desert, part of Arabia, the desert of Gobi, and portions of Mexico, Chili, and Peru it has seldom been known to rain. Coles-Finch states that it seems to be a fact that the atmosphere of the earth is growing drier. The glaciers are retreating, the Caspian Sea and many other lakes are growing smaller, and the great deserts seem to be extending. Some of the richest countries on earth have seen their fertility decreasing, mainly owing to lessened rainfall, and this caused, at least in part, by the ruthless destruction of the forests. Ruined forests mean flooded rivers, periodic droughts, eroded soil, and dried-up springs.

The amount of water given by rain can easily be calculated if two points are known—the mass of rainfall and the area of the receiving surface. The amount is determined by a rain gage and the area of the receiving surface must be measured. Roughly, the amount may be calculated by multiplying the area of the receiving surface in square feet by half the rainfall in inches, the result being in gallons. Here the error is about 4 per cent. Thus, according to Church, one inch of rain on a house roof 20x20 feet area would be about 250 gallons. With a rainfall of 40 inches per annum this would amount to 10,000 gallons, or 27 gallons per day.

The total theoretical amount, however, is never available, for the reason that some is lost by evaporation and the first flow should be wasted. Only a very small proportion of water may be collected from a light shower spread over a considerable interval, especially in hot weather, as nearly all is lost by evaporation.

The stations of Prussia allow the following average for evaporation, the amount evaporated in the open fallow field being called 100:

	Evaporated	Retained More than in Open Fallow Field
	Per Cent.	Per Cent.
Under beech growth	40.4	59.6
Under spruce growth	45.3	54.7
Under pine growth	41.8	58.2
From cultivated field	90.3	9.7

It is this protection against evaporation which gives to the forest its chief value as a guardian of water supply. The forest floor, with its irregularities and its sponge-like qualities, moreover stops the rapid and ruinous draining of the surface, with attendant denuding of the land, and favors slow percolation through the soil and reinforcement of the springs.

The amount of water that can be utilized from the rainfall, draining a catchment area, may be stated as follows: Taking, for example, an average of 46 inches of rainfall each year upon the catchment area, one-half of this is lost by evaporation from the water surfaces, from the surface of the ground, and especially from the leaves of all the plants and trees that grow upon it. The other half, equal to a rainfall of 23 inches, flows off into streams, and sooner or later reaches the lake or impounding reservoir. In wet years the amount that flows off is greater; in dry years it is less than the average; in the winter and spring months the flow is very much greater than at other times.

Collection and Storage.—The points of prime importance in the collection and storage of rain water for domestic purposes are: (1) the material and care of the surface from which it is caught; (2) the separation of the first flow, which contains most of the grossest impurities; (3) the location and construction of the storage cistern.

Storage cisterns for collecting rain water are frequently placed underground. In some places, such as New Orleans, rain water cisterns are built of cypress wood and always above ground. Tanks of wood serve their purpose well, provided they be kept full. If there is great fluctuation in the water line the tank itself falls out of repair. Rain water attacks iron, lead, zinc, and other metals, and when metal cisterns are used the metal should be coated with a good asphaltum paint. This applies also to the delivery pipe. Under no circumstances should lead cisterns or lead service pipes carry rain water used for drinking purposes. It should not be forgotten that cisterns are liable to the grossest kinds of pollution, and they require frequent inspection and cleansing.

Where overflow pipes from rain water tanks are connected with drains precautions must be taken to prevent sewage backing up and entering the tank.

Composition.—Rain water varies in composition with the purity of the atmosphere through which it has passed. It always contains dissolved gases, an average of 25 c. c. per liter. These gases are mainly nitrogen, oxygen, and carbon dioxid, taken up in proportion to their absorption coefficients, and not in proportion to the amount contained in the atmosphere. The gases contained in rain water consist of about 64 per cent. nitrogen, 34 per cent. oxygen, and 2 per cent. carbon dioxid. In addition ammonia is very commonly present. The amount of total solids varies; throughout England it averages 0.39 part per million. The principal inorganic constituent of rain water is sodium chlorid; nitric acid and nitrates, sulphuric acid and sulphate; a small quantity of nitrogenous organic matter is also present. The sodium chlorid comes mostly from the sea spray lifted into the atmosphere through wind action. The sulphuric acid comes

largely from the waste products of burning coal. Rain water is soft on account of the absence of the alkaline earths, and is almost always acid in reaction. It has a mawkish taste.

Bacteria.—Rain water contains a variable number of bacteria and other microorganisms, the number and kind depending upon the germ population of the atmosphere through which the rain passes. Fortunately the various microorganisms floating in the air and carried down mostly by the first shower are not of serious moment, as far as health is concerned. Pathogenic microorganisms in the air are few in number, and these are soon killed by desiccation or the germicidal action of the direct sunlight, to which they are so thoroughly exposed.

Miquel, at the Montsouris Observatory in Paris, found rain water to contain bacteria, pollen, spores of fungi, protococci, etc., especially numerous in the warmer months. In the first showers after a long spell of dry weather over 100,000 such organisms may occur in a pint.

SURFACE WATERS

Surface waters include rivers, creeks, and smaller streams, large and small lakes, ponds, and impounding reservoirs, all resting upon the bosom of the earth in contact with the atmosphere. Surface waters vary greatly in composition, depending largely upon the character of the catchment basin. A water flowing over a rocky soil or through deep layers of sand and gravel is more likely to be free of organic impurities than one that is drained over loam or has stood in swamps.

From the way in which surface waters are exposed they are subject to impurities, and from a sanitary standpoint are frequently dangerous and almost always open to suspicion. Most cities, especially in America, depend upon surface waters for their supply. This is usually taken from rivers, lakes, or impounding reservoirs. It is scarcely possible, in a populous country, to obtain a large quantity of surface water free from pollution with human wastes. Sanitarians have, therefore, more and more come to the conclusion that, while surface waters used for drinking purposes should be guarded against contamination, as far as practicable, they should always be purified before they are used.

Rivers.—Streams are the natural sewers of the regions they drain, and, when used as a source of water supply, we have established a direct connection between the alimentary canals of the people living upstream with the mouths of those below. Most of our large rivers flow through more than one state; therefore, the interstate pollution of streams becomes a national problem. In the older countries of Europe, with more centralized power, laws to prevent the pollution of streams are enforced. In our country the federal authorities are not authorized to enforce this pressing sanitary problem of growing importance. This is discussed more in detail under Sewage.

In our country the rivers furnish the chief source of water supply for most of our large cities. The succession of cities and the combined use of the river as a sewer and source of water supply on such rivers as the Merrimac, Hudson, Delaware, Ohio, Missouri, and Mississippi are particularly impressive, and when the water has been used in its raw or unpurified state much unnecessary sickness has resulted and thousands of lives have been lost in this way.

No stream draining an inhabited region can be considered safe without some method of purification. There are a thousand minor sources of pollution that practically cannot be stopped, even though the sewage flowing into the stream is treated and all reasonable precautions taken in connection with it. It is well known that very few sewage purification works treat all the sewage from the districts which they serve. Thus, there are storm overflows and the street wash that cannot pass through sewers, and other sources of pollution.

Looking at the whole matter of streams pollution solely as an economic engineering problem, it is cheaper to purify the water supplies taken from the rivers than to purify the sewage before it is discharged into them. The volume to be handled is less and the cost of purifying water per million gallons is much less than the cost of purifying sewage. Further, in the present state of our knowledge water may be purified more effectively and with greater certainty than sewage. On the other hand, it is perfectly clear to the sanitarian that the future will require both methods, that is, a reasonable protection of our streams against pollution and the purification of the water served to cities.

Composition.—The composition of river water varies very much, according to the part of the river whence it is taken. Near its source the water may be comparatively pure, but it soon becomes polluted. The composition is complex, as the water of rivers consists of a mixture of rain water and ground water, to which are added surface impurities. As a rule, river water is softer than ground water, but contains a greater amount of organic matter.

Sudden and great changes in the character of river water are to be expected. Other changes, slow in operation but serious in result, come from the increasing pollution with sewage from a growing population upon the upper regions of the watershed.

Rivers are generally purer near their source. The amount of impurities increases as we descend the stream, since the water courses are the natural drainage channels of the country, and the wastes of human life and occupation as well as the scourings of the land find their way into the streams. It is for this reason that rivers, after passing through cultivated valleys with cities, towns, or settlements along their banks, often contain a very great amount of mineral and organic matter.

Thus, the Mississippi at Minnesota contains only 18.6 total solids per 100,000, while the same river at St. Louis contains 244.3 per 100,000.

The amount of mineral matter picked up by a stream depends largely on the geological formation of the country and the erosive power of the stream.

Frequent attempts have been made to correlate the flow of streams and the stages of the river with the outbreaks of disease, especially typhoid fever. It is to be remembered that the flow of streams is dependent in most cases not only on the rainfall, but on springs of local origin. Typhoid may be, and usually is, independent of the stage of the river. Outbreaks are often connected with sudden freshets following a long dry spell, and the explanation seems to be that the accumulated filth is thereby washed down from the slopes and banks of the stream. When streams are very low the flow becomes sluggish, sedimentation and other factors influencing self-purification take place in comparatively short distances; when the river is high the rapid flow is more apt to bring fresh and virulent infection. The decline of typhoid fever in Alleghany in 1908 and 1909 was coincident with an exceptionally low stage of the river. During the spring and fall freshets, when the water is cold and the current swift, the danger is the greatest. In other words, it is the rapidity of flow or the time consumed rather than the stage of the river or the dilution, that is most often responsible for typhoid and other infections in river waters.

If typhoid bacilli are discharged into a stream which flows at a rate of 5 miles an hour, which is a comparatively quiet stream, and accepting the usual figures that the bacteria may die in 5 days, these organisms could be carried 600 miles, surely far enough to reach some domestic supply. Hence, it may be concluded that any pollution, however remote, is apt to reach some consumer unless it occurs near the sea. Nevertheless, the Potomac River at Washington seems to be responsible for little or none of the typhoid fever in that city, although it drains an area of about 11,400 square miles, having a population in 1900 of about half a million and receiving directly the sewage of some 45,000 persons. The question of the self-purification of streams is considered on page 777.

Lakes and Ponds.—Fresh water lakes and ponds make admirable sources of water supply when kept free from pollution with the wastes of human life and industry. This is much more practical than in the case of rivers, on account of the limited area of the catchment basin directly draining into a small lake or pond. Lake water is apt to be soft and free from serious organic impurities. In large lakes the dilution of accidental contamination is enormous, and the effects of time, storage, sedimentation, and other purifying factors have a good chance of exerting their maximum influence. The problem from a sanitary

standpoint is quite different when we consider large bodies of fresh water, such as our Great Lakes, or smaller lakes and ponds.

The Great Lakes.—The lake cities suffer most from the mingling of their own sewage with their own water supplies. This is avoided in part by building the intakes farther out into the lake or by placing the intakes in deep water at points where there seem to be fairly definite currents, bringing fresh, clear water from the body of the lake to the intake. The currents are never constant, being controlled by the wind, hence safety cannot be secured in this way. Almost every lake city has at one time or another suffered from outbreaks of typhoid fever. Chicago has cut a drainage canal to keep her sewage from entering the lake, so that it now flows through tributaries to the Mississippi River. This sanitary reform cost the city of Chicago upward of $40,000,000, and it eliminates the sewage of a large part of the city, but not including certain areas of Evanston and the north side. Despite this commendable piece of sanitary engineering designed to keep the water clean, it is probable that in time Chicago will resort to some method of purifying its water supply. This applies with equal force to all lake cities similarly situated.

Hazen points out that in the smaller cities upon the lakes the mingling of the sewage and water may be relatively just as important as in the larger ones. They have less money to spend, their intakes do not go out so far, their sewers are apt to discharge at the nearest point, sometimes directly in front of the waterworks intake. The water may be shallow and stirred by the wind to the bottom, and, in short, "Menominee's sewage in Menominee's water may be just as bad as Chicago sewage in Chicago water."

The Great Lakes are so large and the dilution and time intervals and exposure to sun and air are so great that there is practically no chance of infection being carried from one of the great cities to another. Thus, Chicago sewage would scarcely endanger the purity of Detroit's water supply, even with no drainage canal. The little city of St. Clair, with 2,543 inhabitants, only 45 miles away, is far more dangerous to Detroit. In the same way Detroit's sewage is probably harmless at Cleveland, and Cleveland sewage is harmless at Buffalo. The sewage of Buffalo, however, is a great menace to those drinking the water at Niagara.

Impounding Reservoirs.—Impounding reservoirs are artificial ponds or lakes, usually made by throwing a dam across a narrow valley. Most impounding reservoirs are made along the course of a small stream.

The principal use of impounding reservoirs is to hold the excess of water of the winter and spring flows and make it available during the summer and fall.

The impounding reservoir designed to furnish New York City with a new supply of water to supplement the Croton system will be the largest artificial reservoir for water supply in America, if not in the world. It is situated in the Catskill mountains, and is made by damming Esopus Creek, and will ultimately hold one hundred and twenty billion gallons of water. Boston is supplied from impounding reservoirs on small streams; the Cochituate (1848), the Sudbury (1878), and the Nashua (1898). The Wachusett reservoir stores the combined water from the smaller sources of supply, and has a capacity of 63,000,000,000 gallons of water. Baltimore has an impounding reservoir upon the Gunpowder River; other cities similarly supplied are Newark and Jersey City in New Jersey; Worcester, Cambridge, and Springfield in Massachusetts; New Haven and Hartford in Connecticut; Altoona in Pennsylvania, and Denver in Colorado; San Francisco and Oakland in California; and numerous other smaller cities. From a sanitary standpoint the great advantage of an impounding reservoir is that it drains a comparatively small area that is amenable to control; often the catchment area is in uninhabited hilly or mountainous districts. The other sanitary advantage lies in the fact that benefit is taken of the great sanitary safeguard of storage. Most pathogenic microorganisms die a natural death during the time that the water is stored in a large impounding reservoir. In Boston it is estimated that the water is stored an average of 30 days before it reaches the consumer. Few non-sporulating bacteria dangerous to man can live so long in water under natural conditions.

The chief disadvantage of impounding reservoirs as storage basins is that they are open to the air and light, and thus favor the growth of algæ and other microscopic organisms responsible for objectionable tastes and odors. Further, the stagnation of the water favors the accumulation of the products of decomposition, which is another source of evil smells and vile tastes. The stagnation of water in impounding reservoirs and small lakes and ponds deserves special mention.

Stagnation of Water in Impounding Reservoirs and Small Lakes.—Hazen points out that in our climate, when a reservoir or lake is more than 20 to 40 feet deep, the upper part of the water is usually in circulation under the influence of the wind, and the lower part remains stagnant. There is little or no mixing between the surface water and the bottom water, except for two short periods each year, one in the spring and one in the fall. These periods of circulation to the bottom are known to waterworks men as the spring turnover and the fall turnover.

During the summer weather a blanket of warm and, hence, light water remains at the surface. This layer may be 20 feet in small reservoirs, and 40 feet in great lakes. The temperature of this surface

layer may reach 75° or 80° F. or more in midsummer. The wind stirs it up to a certain depth (about 20 to 40 feet), depending upon the depth of the reservoir and the force, direction, etc., of the winds.

The bottom layer is cool and quiet. As the air temperature falls with the approach of winter the surface water cools, until it approaches that of the bottom water. When the difference in temperature between the surface and bottom layers is less, the wind action extends deeper, until, all at once, often when the wind is blowing, vertical currents arise, so that all the water in the reservoir turns over and mixes from top to bottom. The mixing continues for a few weeks, until the temperature of the surface water falls below the point of maximum density, namely, 4° C. Then the colder water commences to accumulate at the top. The top often freezes and entirely shuts out wind action, so that the period of winter stagnation is even more quiet than the summer period. The spring turnover is caused by a reversal of the conditions causing the fall turnover; surface water is warmed until it reaches the temperature of the bottom water, when the upward and downward currents take place.

It can readily be seen that this phenomenon has much to do with the quality of the water. Thus, the organic matter upon the bottom of almost all reservoirs decomposes, and in the absence of oxygen produces the vile odors and nasty tastes of putrefaction. These odors and tastes accumulate in the bottom water until the fall turnover; then they become mixed with all the water in the reservoir. If the water is drawn from the reservoir near the top, as it usually is, there will be a great change in the quality of the water on the day of the fall turnover. The surface water is well charged with oxygen, and, as this falls to the bottom, it oxidizes and neutralizes some of these products of decomposition. Tastes and odors due to this cause may be removed by aerating the water by means of fountains, cascades, falling over a dam, or any other similar means. For a further discussion of this interesting subject see Hazen's "Clean Water and How to Get It."

Stripping.—Stripping consists in removing the organic matter of the surface soil, which is to become the bed of a reservoir. The object of stripping is to diminish the amount of putrefaction taking place in the bottom stagnant water, and also to furnish less food for bacteria and algæ. A number of the reservoirs in Massachusetts were first stripped at considerable expense. It has been found that in the older reservoirs prepared in this way putrefaction has not taken place for some years, although in some cases putrefaction seems not to have been entirely prevented, even at the outset. Stripping does not prevent objectionable growths; it only reduces them somewhat, because many of the organisms do not need or make use of the organic matter of the soil as their food supply. The algæ live rather on the mineral mat-

ters of the water and the air, and, with the aid of the sunshine, they build up their own organic matter, precisely as the higher plants do growing in soil.

GROUND WATER

Water which is taken from the ground by means of wells or flowing naturally from the ground, as in springs, is usually satisfactory, as far as injurious impurities are concerned. The surface water is greatly purified as it percolates through soil. This is nature's process of filtration; the organic matter is oxidized, the bacteria are largely strained out. The soil can take care of a large amount of pollution, and, if not overburdened, or if it has no cracks or crevices, the ground water may be entirely free of objectionable organic substances and bacteria. In passing through the soil the water takes up a rather large amount of carbon dioxid, which is set free from organic decomposition. The water, thus acidulated, has a greater solvent action for lime and other mineral constituents, so that ground water is apt to be harder than surface waters, and to contain a larger amount of dissolved inorganic substances. In deeper waters the solvent action is favored by increased heat and pressure, so that deep wells and artesian waters are frequently unfit for domestic use on account of the large amount of inorganic impurities which they contain, such as lime, iron, common salt, etc.

The water that soaks into the soil finally rests upon an impervious stratum. Such water, as a rule, does not exist in the ground as a river or lake, but occupies rather the spaces between the sandy particles, except in limestone formations. Ground water, therefore, in any quantity is found, as a rule, in sandy, gravelly, or sandstone formations.

It is only in limestone regions that the ground water exists as flowing rivers or in large bodies. In such instances, as, for example, the mammoth cave in Kentucky, the underground river may appear and disappear suddenly. The sanitary significance of water from limestone crevices is entirely different from that obtained from a sandy soil.

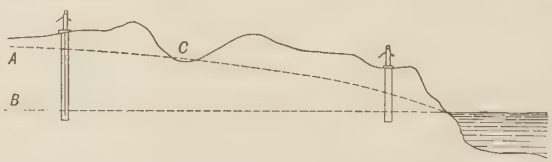


FIG. 94.—GROUND WATER. A. High level. B. Low level. C. Intermittent spring.

The surface of the ground water does not follow the surface of the land, but more approximately the contour of the impervious stratum on which it rests. It crops out at the surface here and there, to form rivers, ponds, lakes, and springs. The irregularity of the surface of the ground water table is due to a certain extent to the rainfall. During

drought the level becomes more and more uniform, until it may become quite horizontal. In most cases, except where water lies in deep depressions and pockets, the ground water is in constant lateral motion. This motion is usually in the direction of outfall, that is, toward the nearest large body of water—lake, river, or sea. That is why fresh water may frequently be obtained by sinking a well at the seacoast. In some places the rate of lateral flow is so slow as to be almost imperceptible; at other places it is comparatively rapid. Thus, at Munich, Pettenkofer estimated 15 feet per day; at Budapest, Fedor found the ground water to flow at an average of 167.6 feet per day. The rate of flow of ground water may be determined approximately by several methods.

The method of determining the velocity of ground water which has been used with satisfactory results by Thiem is as follows:

Three or four borings are sunk to ground water in a line in the direction of flow. A large dose of salt is then put into the upper hole, and at frequent intervals analyses are made of water drawn from each hole below, until the salt content has reached its maximum in each case, and the rate of movement is computed from these results.

Amount.—The amount of water that may be obtained from the ground can only be determined by means of actual pumping tests carried on for a sufficient length of time to bring about an approximate state of equilibrium between the supply and the demand, as determined by the level of the ground water. It is rarely practical to continue such tests until perfect equilibrium is reached, for in many cases several years of operation would be required to determine the ultimate capacity of a source. Pumping tests of short duration are apt to be very deceptive, as ground water may exist in the form of a large basin or reservoir with very little movement, corresponding to a surface pond with small watershed, and brief tests would give little more information than similar tests on a pond.

It is easier in proportion to get a little ground water than to get a large amount, and for this reason ground water supplies are more generally available for, and better adapted to, the needs of small places than of large cities.

In Europe, ground water supplies have been secured for many large cities; there has been no corresponding development in America. The reasons for the greater use of this method of supply in Europe are: smaller quantity of water required per capita, more favorable geological conditions, and more study given to the subject and greater efforts to secure them, especially in Germany.

Ground water may be obtained from: (1) sand and gravel deposits, (2) sandstone rock, (3) limestone formations.

Ground Water from Sand and Gravel Deposits.—Water flows through sand with some difficulty. From a given pumping station it

is only possible to draw the water for a limited distance. This distance depends upon the depth and coarseness of the sand. Therefore, the only way to secure a large quantity of water from such formations is by the use of a number of comparatively small pumping stations, separated so as not to draw from the same territory.

Only a given amount of water can be secured from a square mile of ground. The amount depends upon the rainfall, upon the evaporation from the surface of the ground from transpiration of vegetation, and upon the amount of storage in the pores of the soil.

Most of the sand deposits of our country are not practically available for water supply purposes, because the grains of sand are too small and the flow of water through them is too slow. It is only the coarse-grained sands that are practically available.

A few cities in America obtain their drinking water supplies from ground water obtained from sand and gravel deposits. At Brooklyn the conditions are particularly favorable, and it is estimated that 78 million gallons of ground water are obtained each day for that city. For this purpose 24 separate pumping stations are used. The water supplied to Camden, N. J., is obtained from the ground through wells close to the Delaware River, and the amount is increased by taking river water from the surface of some of the ground about the wells. This water filters through the sand slowly and is well purified. This method of adding to the yield of wells is used in some places in Germany and France. Memphis, Tenn., is probably the largest city in the United States supplied entirely with water drawn from sand and gravel deposits. In this case the water-bearing area is several hundred feet below the surface, and is below a clay layer. Lowell, Mass., obtains ground water from three stations, draining different areas of glacial drift.

Filter galleries or excavations in sandy materials near river banks have been used in the past. Such water corresponds in all practical respects to the ground water obtained from sand and gravel deposits by means of wells. The wells are preferable, as they allow water to be drawn at a lower level, and this tends to a drainage of a greater area, thereby securing a larger quantity of water.

Filter galleries are apt to furnish a diminishing supply, because the pores of the filtering material become filled with the sediment of the river water. When this happens there is no way of renewing the source. In some torrential streams the filtering surface is renewed from time to time, but this usually does not occur.

Ground water obtained from sand and gravel deposits is usually clean and free from unwholesome impurities. Nevertheless, many towns and cities which were formerly supplied with such water were compelled to seek other sources, because sufficient water was not obtainable

from the ground to supply the increasing quantities required by rapidly growing population.

Ground Water from Sandstone Rock.—The method of driving wells in sandstone rock differs from that in driving wells in sand or gravel, but the collection, storage, and flow of water are precisely the same.

The cementing material, which binds what otherwise would be loose sand into a solid rock, often seems to offer but little resistance to the flow of water, and the sandstone for water supply purposes acts as so much sand would act.

Water drawn from sandstone is always well filtered. It, however, is usually limited in amount, and, while of the greatest value for small supplies, is not available for large communities.

The Marshall and Potsdam sandstone underlying parts of Michigan, Illinois, Wisconsin, and Minnesota are used extensively for supplying towns and small cities. Thus, Jackson, Mich., with a population of over 25,000, is one of the largest cities so supplied.

Ground Water from Limestone Formations.—In limestone formations the underground flow of the water is not through sandy or porous rock, for limestone is not porous. The water travels through fissures or passages. When these are large they are called caverns or caves, as, for example, the mammoth cave in Kentucky. These caverns or caves are natural seams or cracks enlarged by the gradual solution and removal of the limestone by the passing water. Limestone is the only common rock that is soluble in this way, and, for water supply purposes, limestone formations must be distinguished from all others.

The crevices may be, and often are, continuous for many miles. They are remarkably tortuous and anastomose freely, and the direction and flow of the water bear no relation whatever to the surface topography. Pollution at one point may, therefore, endanger those using the water at a far distant place.

Limestone formation has little ability to hold the abundant winter flows to maintain a supply through droughts. The difference between limestone and sand in this respect is striking, and, from a sanitary standpoint, the fact that water flowing through sand is filtered and purified, whereas no such action takes place through limestone fissures, is significant. While much water is frequently available at one point in limestone formations, the amount is subject to greater fluctuations, and the supply may fall short when most needed.

That contamination at one point may soon reappear at a far distant point may be demonstrated by the use of fluorescent dyes, or by the use of massive cultures of some harmless microorganism, such as yeast or *Bacillus prodigiosus.*

In our country San Antonio, Texas, is supplied with water from limestone springs flowing in greater volume. Indianapolis was at one time and Winnipeg in Canada is still supplied largely from this source. Paris in France is partially supplied with limestone water. Vienna obtains its supply from the wonderful Kaiserbrunnen and other limestone sources, which are all in the high mountains, where there is scarcely any population or pollution. This supply is mainly from the melting ice and snow of the high mountains which replenishes the springs, so

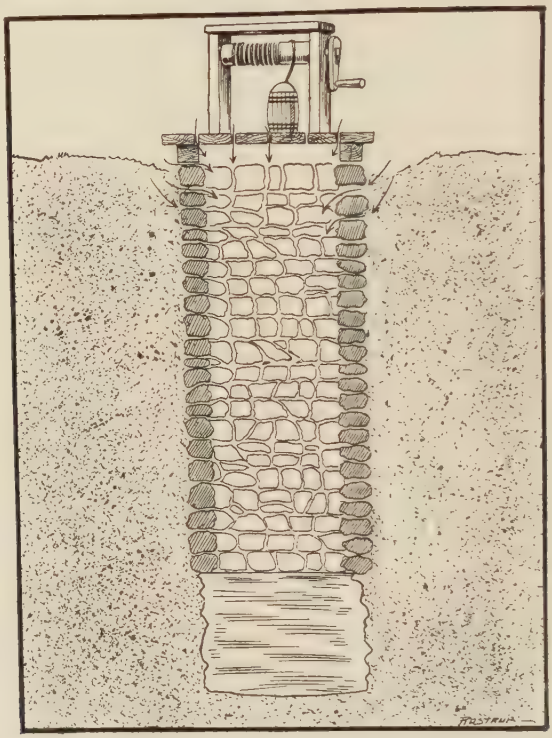


FIG. 95.—USUAL METHOD OF POLLUTION AND EVEN INFECTION OF WELLS.

that the amount of water obtainable is greater in summer than winter.

Typhoid fever has been caused rather frequently by the use of ground water from limestone formations. This has been demonstrated in Paris, Switzerland, France, and England. Water supplies from limestone formations must, therefore, be regarded with suspicion.

Wells.—A well is nothing more or less than a hole sunk into the earth to reach a supply of water. Wells may be either shallow or deep, dug or bored. By a shallow well is usually understood one which is dug and lined with stone or brickwork. The cylinder is usually 5 or 6 feet in diameter and rarely over 30 feet deep. By deep wells are meant drilled or the so-called artesian wells. They consist of an iron pipe or

tube 6 to 8 inches in diameter, and may extend many hundred feet into the earth. If the water is drawn from a depth of 100 feet or more without passing an impervious stratum, the well is usually spoken of as a deep well. If the well passes through an impervious stratum into a pervious one beneath, in which the water rests upon another impervious stratum, it is spoken of as an artesian well. Water is usually pumped from the wells either by means of the ordinary suction pump or by means of compressed air.

Contrary to the generally accepted opinion wells are usually polluted from the surface and not from the sub-soil drainage. The filtering power of the soil is usually sufficient to protect the water drawn from a well, unless (1) the soil is overburdened with organic matter, or (2) a cesspool, broken sewer, or other gross source of pollution is very close, or (3) channels, fissures, or crevices exist in the soil and subsoil so that impurities reach the well without undergoing the process of biologic filtration.

In locating a well, therefore, much depends upon the surface configuration of the ground, the character of the soil, and the proximity of possible sources of pollution. The casing of the well should be sound and tight, preferably of brick laid in cement mortar, pointed on the inside. This impervious casing should extend as deeply into the well as practicable, and after it is laid the outer space between the casing and the earth should be filled in with well-tamped clay soil. One of the most important points in the construction of a shallow well is to extend the casing at least 18 inches above the surface of the ground and to build around it a shield of concrete or brick laid in cement extending in a circle from the top of the well 3 or 4 feet wide. This shield should join the well casing so as to make a tight joint with the casing. The floor of the well should rest upon the top of the casing, so that no space is left for frogs, mice, or bugs to crawl in. The floor should likewise be water-tight, and is best made of reinforced concrete with a cement surface. If this is not practicable, it should be made of sound, hard, tongue-and-grooved boards well driven up, and the edges painted with white lead. Upon this should be laid another floor of similar material at right angles to the first. The pump should be let into the floor and firmly fastened to it, and

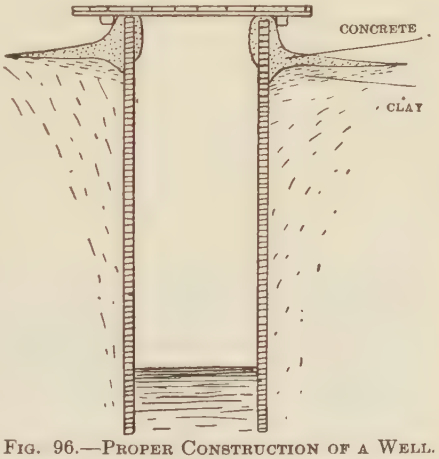


FIG. 96.—PROPER CONSTRUCTION OF A WELL.

protected with a flashing of tin to prevent water washing back into the well.

The widely prevalent idea that some form of ventilation must be provided for a well is entirely unnecessary. Well water keeps better in the dark and protected from the outer air.

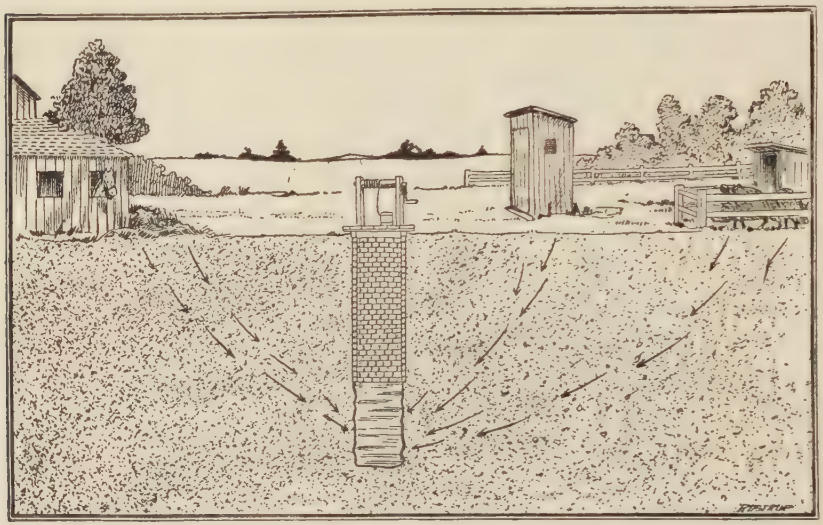


FIG. 97.—POPULAR IDEA OF HOW WELLS BECOME INFECTED FROM SURFACE POLLUTION. This probably rarely takes place in rural districts, as the soil can usually hold back most of the impurities. The danger is great, however, where fissures, cracks, or crevices exist, or where sewage enters beneath the surface of the soil from broken drains or leaky privies.

The top of driven wells should be as carefully protected as those just described for a dug well, as otherwise the polluted surface water may work down the sides of the pipe. Care should be taken that the pipes of a driven well near the surface of the ground do not rust and become leaky. Such wells should be provided with a heavy top, to which the pump frame should be tightly bolted, in order to prevent the loosening of the joints in the pipe by the vibration of pumping. The ground about all wells should be kept clean, and, where possible, should be turfed. The waste water should be carried by pipes to a considerable distance from the well.

Artesian water and water from deep wells furnish the safest and most satisfactory sources of supply we have. Such water is usually clear and of high sanitary quality. Sometimes such waters contain a large amount of inorganic impurities, which render them unfit for domestic purposes. Frequently they contain iron in the ferrous state, which soon oxidizes upon contact with the air and is thrown out as an insoluble ferric salt, which renders the water yellowish or brownish.

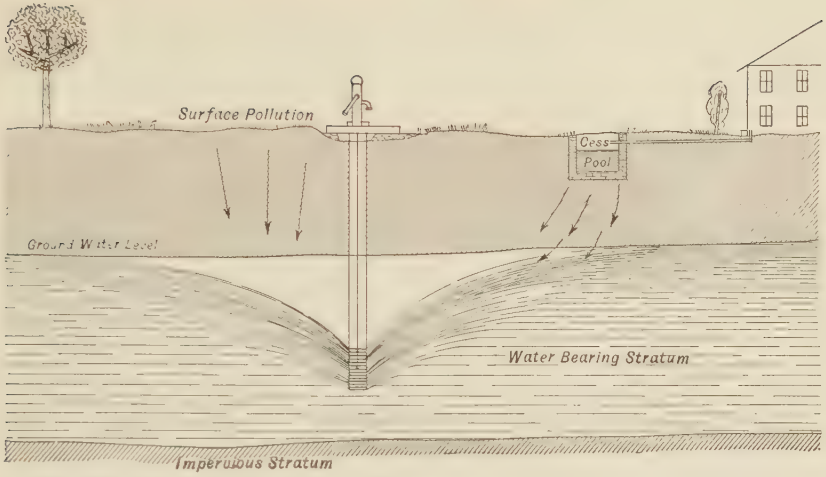


FIG. 98.—DEPRESSION OF THE GROUND WATER LEVEL BY PUMPING AND TENDENCY TO DRAW NEARBY POLLUTION FROM THE SOIL OR CESSPOOL.

Deep well waters may also contain an excess of lime salts or common salt.

Water from shallow wells obtained from sandy or gravelly formations are entirely satisfactory, provided there are no nearby sources of pollution. The proximity of well and privy may be especially hazardous. Shallow wells in limestone regions must be carefully guarded and always looked upon with suspicion.

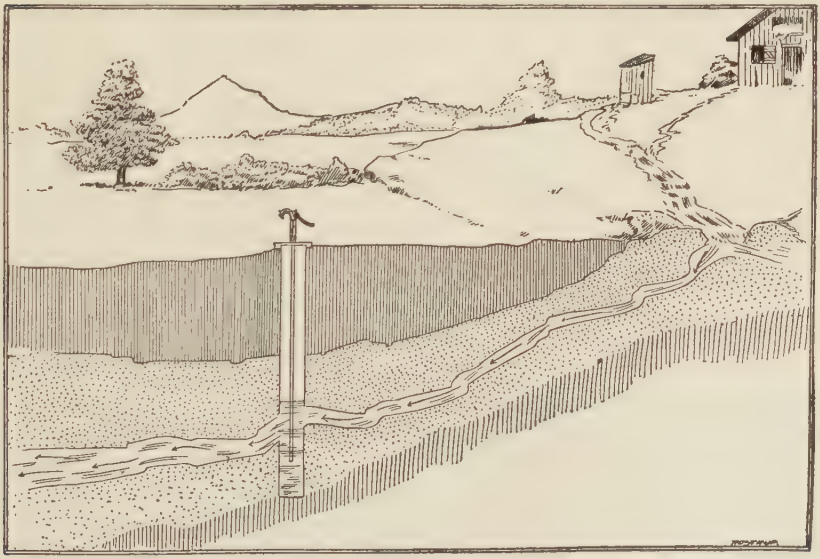


FIG. 99.—IN A LIMESTONE FORMATION IT IS DIFFICULT TO TELL ANYTHING ABOUT THE SOURCE OF WATER OBTAINED FROM A WELL.

It is evident that in a densely inhabited area with miles of sewers, some of them doubtless broken or leaky, and with the thousands of privy vaults which still survive in most of our American cities, we have a more or less sewage-polluted condition of the soil favorable for the contamination of shallow wells. Shallow wells, on general principles, have been gradually eliminated from all large cities having an abundant water supply. This danger was well shown in the studies upon typhoid fever in the District of Columbia, in which many of the shallow wells situated within the city limits were shown to be polluted by chemical and bacteriological analyses.

Wells may be disinfected with lime, which has been found to be fairly effective. A mixture of carbolic acid and sulphuric acid in sufficient quantity will sterilize a well, but these substances have evident objections. The method of injecting steam under a pressure of two atmospheres has been used. The steam is forced into the water until the temperature is brought to near the boiling point. Bleaching powder, however, is the cheapest and most practical method of disinfecting wells that need such purification.

Springs.—Spring water does not differ in any essential particular from the ground water obtained from shallow wells. Springs may be regarded as natural wells, outcropping where the geological formation is favorable. Spring water, as a rule, is of a high degree of purity, and as the water flows spontaneously it can easily be utilized; and, as no form of machinery is necessary to pump it, it is less subject to contamination than well water. Spring waters differ greatly in character, depending upon the temperature of the water and the inorganic constituents which it contains. Springs may be perennial, the flow being constant or intermittent.

Springs may be polluted from various sources. The overlying porous layer of soil may be too thin to remove the contamination of surface washings from privies, stables, hog pens, and other sources of contamination. This is probably not a frequent source of danger in such waters. Springs may be contaminated from surface washings; that is, the infective material may be washed down and into the spring by heavy rains, and, unless the spring has a bold flow, the polluting material may remain in it for some time. Leaky cesspools above a spring may carry dangerous material almost directly into the water, just as they endanger wells in precisely the same way.

The protection of a spring against contamination requires a careful study of each location. Stables, hog pens, and privies should be distant, and, if possible, on another slope. Soil pollution must be prevented in the neighborhood of the spring, and animals kept away, and special regard must be had for the location and character of the privy. The spring should be protected above with a masonry or concrete wall. This

should extend well into the ground, so as to guard against surface washings. A ditch should be dug to carry off the surface water around both sides of the spring, and the neighborhood kept clear of weeds and growth. It is well to plant grass about the spring so as to keep out dust and prevent erosion of the soil.

FIG. 100.—SPRING (ON THE LEFT) EXPOSED TO CONTAMINATION FROM SURFACE WASHINGS FROM THE HILL ABOVE. SPRING (ON THE RIGHT) PROTECTED FROM SURFACE WASHINGS; THE BUCKET CAN BE FILLED WITHOUT CONTAMINATING THE FLOW. (Virginia Health Bulletin.)

In limestone regions springs are subject to the danger already spoken of in the case of wells. A spring in such a region may be the same underground stream that runs through the neighbor's back yard and disappears in his meadow. A limestone spring that becomes muddy soon after a rain should be regarded as particularly suspicious.

THE SOURCES AND NATURE OF WATER POLLUTION AND INFECTION

A distinction is drawn between a polluted and an infected water. A polluted water is one that contains organic matter and the products of decay, either of vegetable or animal origin. An infected water is one that contains the specific parasites causing disease. A polluted water may not be particularly harmful to health; it is always suspicious. That is, a polluted water is not necessarily infective; an infected water is practically always polluted. Practically all surface waters are pol-

luted; ground waters usually show evidence of past pollution; that is, they contain inorganic salts in solution resulting from the mineralization of organic matter.

The greatest hazard to man is found in a water polluted with the discharges from the human body—feces, urine, and sputum. There is comparatively little danger from water containing the wastes of other animal life, for the reason that few of the infections of the lower animals are thus transmissible to man. There is still less danger in water contaminated with organic matter of plant origin. Water containing inorganic substances in solution plays a relatively minor rôle, as far as health is concerned.

From a sanitary standpoint, then, it is the wastes of human life that concern us especially. These may enter a surface water directly from overhanging privies, or from sewers, or from washings of the land. Ground water becomes polluted in ways already discussed.

The prevention of the pollution of our streams, lakes, ponds, and other surface supplies is an important sanitary problem with a large economic side. As far as streams and large lakes are concerned, the most dangerous infection is that which is nearby—that is, that which is quickly transferred in a fresh and virulent form. Distant infection is much less dangerous. Cities taking water from an average stream should prevent the access of direct pollution for at least 50 miles, or better 100 miles, above the intake. Partial protection may also be accomplished by requiring sewage disposal works for all towns and settlements, and abolishing all overhanging privies upon the river and its tributaries. A sanitary inspection could cover a large shed for this purpose. When these measures are not feasible, intercepting sewers may be built, as on the Schuylkill at Philadelphia. Canals that parallel a river, as the one upon the bank of the Potomac, may receive the sewage and surface drainage and thus protect the stream. It is comparatively easier to guard smaller lakes and ponds and impounding reservoirs.

Simple Tests to Determine Sources of Pollution.—Sources of pollution and possibly of infection may often be determined by simple tests which may be carried out by a layman. These tests afford valuable information and consist in the addition of some chemical substance to the source from which pollution is possible and then determining whether the same reappears in the water supply. For this purpose a large number of substances that may be readily recognized by their taste, odor, or appearance may be used, such as coal oil, carbolic acid, fluorescin, and common salt. Coal oil poured near the ground of an artesian well is an easy and convincing method of establishing the presence of defective piping and surface or sub-soil contamination. Nördlinger recommends for this purpose saprol, which tastes like naphtha and is so penetrating that its odor may be readily recog-

nized in proportions of 1-1,000,000 or by taste in solutions of 1-2,000,000. Trillat experimented with a large number of dyes and finds that fluorescin dissolved in alcohol and diluted with 5 per cent. ammonia solution can be detected by a fluoroscope in proportions of 1-2,000,000,000. The fluoroscope is a tube of clear glass three or four feet long and one-half inch in diameter, closed at one end with a rubber cork. In such a tube natural waters have a somber blue color which changes to a clear green if fluorescin is present. This dye possesses the evident advantage of not being precipitated by the soil ingredients, a reaction that readily occurs with most aniline dyes brought in contact with calcareous solutions. Salts of lithium are sometimes used, for they may be detected in the minutest traces if the water is examined by the aid of a spectroscope.

The conclusion must not be drawn that because these soluble salts reappear in the water microorganisms and dangerous pollution would likewise find its way through the soil for an equal distance, for the soil has well-known filtering power when free from fissures or actual channels and is capable of removing bacteria and oxidizing large quantities of organic matter. However, these methods are of service in indicating the possibility of danger under certain circumstances and are particularly useful in discovering sources of pollution near wells or in limestone formations.

Massive cultures of prodigiosus, yeasts, and other microorganisms if not normally present in the water under examination may be used to detect the possibility of pollution. The cultures are poured upon the ground or into suspicious places and the water tested at varying intervals to determine whether they reach the supply. Careful controls must be made beforehand to assure the absence of the particular organism used.

The Interstate Pollution of Streams.—Sanitarians have maintained for years that no community or individual has a right to pollute streams used for public water supplies, any more than a man has a right to poison his neighbor's well. The legal aspects of water pollution have been carefully considered by Dr. J. L. Leal. England enjoyed the benefit of a Rivers Pollution Commission as early as 1855, in order to prevent, remedy, and remove the danger of polluted water supplies. This commission adopted a comprehensive system for the disposal of sewage and for water purification, the fruits of which England is enjoying to-day. This country has no law regarding the interstate pollution of streams, and with our growing population and increasing amount of pollution this is becoming a live and pressing sanitary question. After the Chicago drainage canal was opened the city of St. Louis (state of Missouri) sued the city of Chicago (state of Illinois) through the federal courts, asking an injunction against the pollution

of the Mississippi River, from which St. Louis draws its drinking supply. The testimony occupied many weeks, and in published form takes up many volumes. The verdict was "no cause for action," or "not guilty," that is, it was not proven that typhoid bacilli or other organisms dangerous to health reached St. Louis from Chicago.

The principles of common law as to interstate waters have been appreciated by some of the nations of Europe. Thus, the inhabitants of a town in Belgium suffered from the effects of a river polluted by the French, and the French government not only compelled the offending city to dispose of its sewage by irrigation, but granted a subsidy for this purpose. In some of our more progressive states, as, for example, Massachusetts, Pennsylvania, Connecticut, Minnesota, New Hampshire, New Jersey, New York, Vermont, and others, the State Board of Health is given control over the pollution of streams within the borders of the state.

Speaking generally, jurisdiction over the pollution of waters in the United States is confined to the several states. There is no provision in the Constitution which gives to Congress authority in the premises. Hence, by the familiar principle in our Constitution that the several states retain full sovereign power, except so far as such powers are restricted by the national constitution or expressly delegated thereby to the national government, the individual states have full control of this subject—a subject with which they are individually impotent to deal and which logically belongs to the federal government.

The Care of Catchment Areas.—The ideal catchment area is free from human habitation and is covered with forests. The catchment areas supplying impounding reservoirs and the natural ponds and lakes used as reservoirs are limited in area when compared, for example, with the catchment areas of the great rivers, from which many public water supplies are drawn. It is, therefore, possible to inspect and control the former more readily than the latter.

It is often impossible to remove population from a catchment area, and, in fact, it is usually unnecessary to do so. Very good water may be drawn from areas upon which there is a large population, when proper and well-known precautions are taken. Thus, there are 776 people per square mile upon the Cochituate catchment area, 282 upon the Sudbury, 49 upon the Wachusett, furnishing Boston's water supply, and 59 upon the Croton, furnishing New York's water supply. The prolonged storage of the water in large reservoirs is a sanitary safeguard, and makes the Boston water and the New York water safer than it otherwise would be.

The greatest danger is that some polluted water will sometimes get by the reservoir or flow through it by some short circuit, and so reach

the consumer, before it is subjected to full storage conditions for a sufficient length of time.

The proper sanitary care of a catchment area requires, first of all, sufficient laws granting suitable authority, especially concerning the disposal of human wastes.

Care must also be exercised to keep out manufacturing wastes and the surface washings that may carry pollution from human sources or undesirable contamination from other sources. This object may be accomplished in various ways. The city should own the shores of the reservoirs and also as much of the land along the important streams as is necessary to carry out these objects. Old sources of pollution must be removed, and new sources not permitted. Where the danger from human pollution is especially great, as around the impounding reservoir itself or at nearby suburban settlements, engineering projects, sometimes of considerable magnitude, are necessary to carry away the sewage and the surface drainage. A strict patrol of the catchment area, in order to supervise picnic and camping parties, the camps of construction gangs, and other sources of danger, must be exercised. A good man on the alert can patrol a large district, getting his information through various ways, and personally inspecting all suspicious localities frequently.

In the investigation of a stream and its watershed the chief points requiring attention are the relative proportions of the polluting matter and the flow of the river when at its minimum; the general character of the stream, the rate of flow, and the distance between the source of pollution and the intake of the water.

Many water boards, having control of large tracts of land, are planting their catchment areas with trees with advantage and profit, for it is found that the presence of trees adds to the retention of water falling as rain as well as by radiation, and cooling the adjacent atmosphere, perhaps aiding condensation and rain. It prevents floods, regulates and helps to purify the supply, for water draining through the soil of wooded areas is naturally cleaner than that scouring the surface of barren land.

CHAPTER II

SANITARY ANALYSIS OF WATER

A complete sanitary analysis of water includes: (1) a *physical examination* to determine color, turbidity, odor, and taste; (2) a *microscopic examination* to determine the number and character of particles in suspension, especially algæ; (3) a *chemical analysis* to determine the nature and amount of chemical impurities; (4) a *bacteriological examination* to estimate the number and kind of bacteria; (5) a *sanitary survey* of the watershed, including the methods of collecting, storing, handling, and distributing the water; and (6) *clinical experience*, which, after all, is the final test, for water may contain impurities that are not recognizable by any other method.

Water is particularly liable to contamination under prevailing conditions and must, of necessity, demand increasing watchfulness and a continual readjustment of restrictions governing its use. Water may contain impurities beyond the power of science to disclose. Thus, the water supply of Vienna from the famous Kaiserbrunnen is particularly pure, as determined by laboratory analysis. Nevertheless, this water supply is said to be responsible for a great increase in the amount of goiter which has occurred in Vienna since its introduction.

The fact that water is the most universal solvent known is not to be neglected. The water we drink has come in contact with the earth and many other substances. It dissolves many organic and many inorganic impurities, few of which can be detected in the laboratory by the routine methods used. The influence of many of these substances upon health is unknown. Exceedingly small amounts of poisonous substances in water may act injuriously when we recall how much water is daily taken. All these facts should make us cautious before we give a water supply a clean bill of health, and communities will find it pays in the end to go to great expense to improve this important article of daily use.

Standard Methods.—The advantages of using a standard method are self-evident; it at least gives results that are fairly comparable with the work of others. The standard methods for water analysis have been carefully considered by a competent committee of the American Public

Health Association. The first report was published in the *Journal of Infectious Diseases*, Supplement No. 1, May, 1905.[1] Amendments and improvements to the method are published from time to time. For anyone not having special skill in chemical analysis or bacteriological technique it is advisable to adhere closely to the standard procedures. Any deviation from these methods should always be noted in published reports. Because a method is "standard" does not mean that it has a fixed and permanent value as a model to be blindly followed under all circumstances. Standard methods are established by common consent as the rule to be followed under ordinary circumstances, especially for routine work and by those who are not especially skilled in laboratory technique. For reasons that seem self-evident, it is of special importance to follow the standard methods for bacterial counts.[2]

Our standards by which the purity of water is judged are constantly rising. There is no doubt that many waters now considered pure and wholesome will not be acceptable in the future.

ODORS AND TASTES

The purest water is absolutely devoid of taste and odor, but it is also insipid. If such water is aerated by agitation or by filtration through a porous air-containing substance, it becomes sparkling and agreeable.

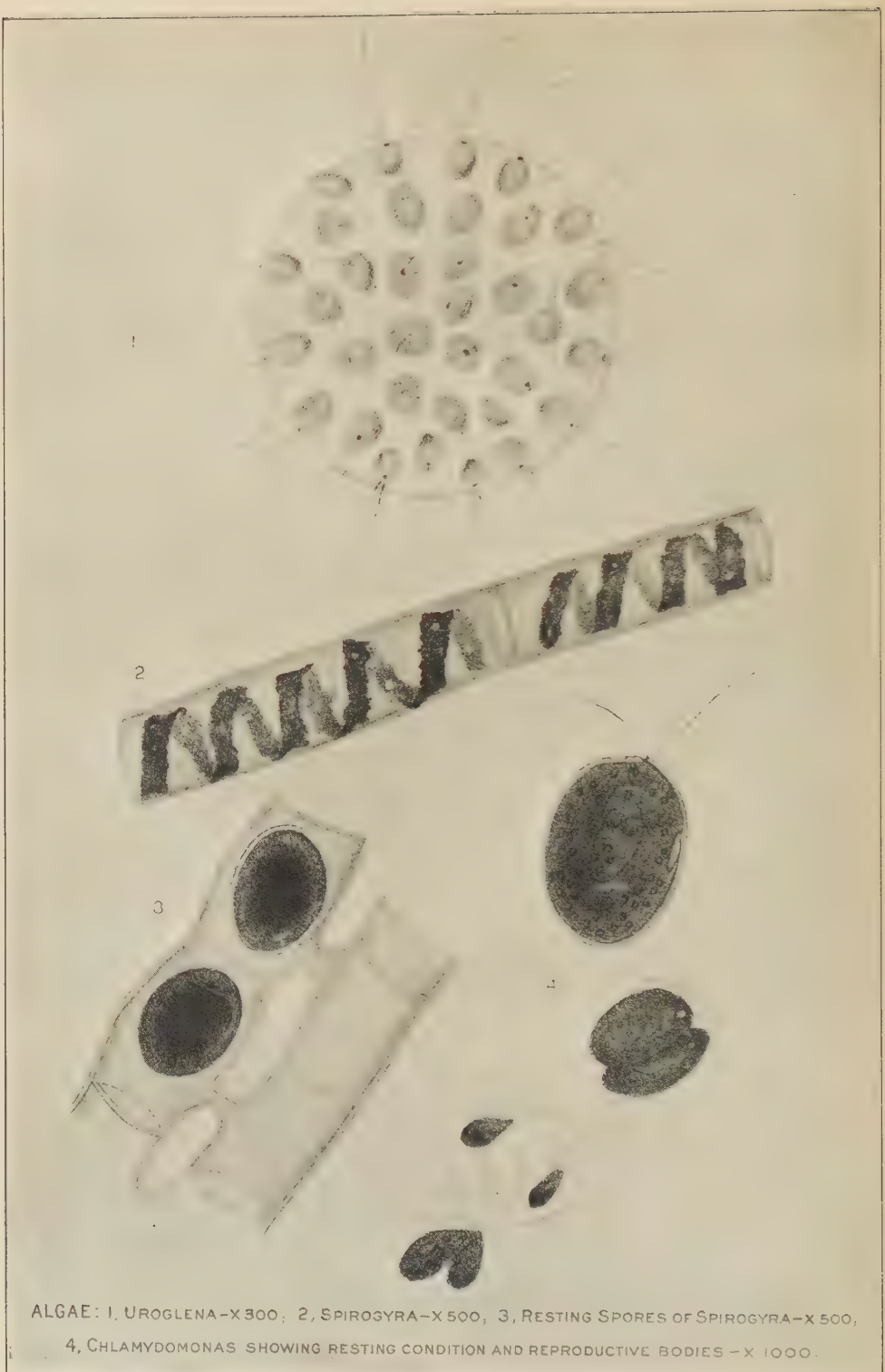
Odors in waters are objectionable, rather than detrimental to health. As a rule, the most objectionable odors develop in surface waters and are caused by the growth of algæ, diatoms, protozoa, and other microscopic beings. The earthy odor of some ground waters is due to substances taken up during the passage of the water through the soil. When a well-water becomes offensive it is evidence of stagnation at the bottom of the well or the presence of dead animals. In the case of deep wells hydrogen sulphid and other inorganic compounds may impart odors to the water. The odors and tastes which develop in impounding reservoirs from stagnation and putrefaction of the organic matter have been discussed on page 706.

On the whole, the waters of natural lakes and ponds are less subject to objectionable odors and tastes than are the waters of artificial reservoirs, and putrefaction is less troublesome, but the difference is one of degree, not of kind.

The power of water to dissolve or absorb gases and odors is an im-

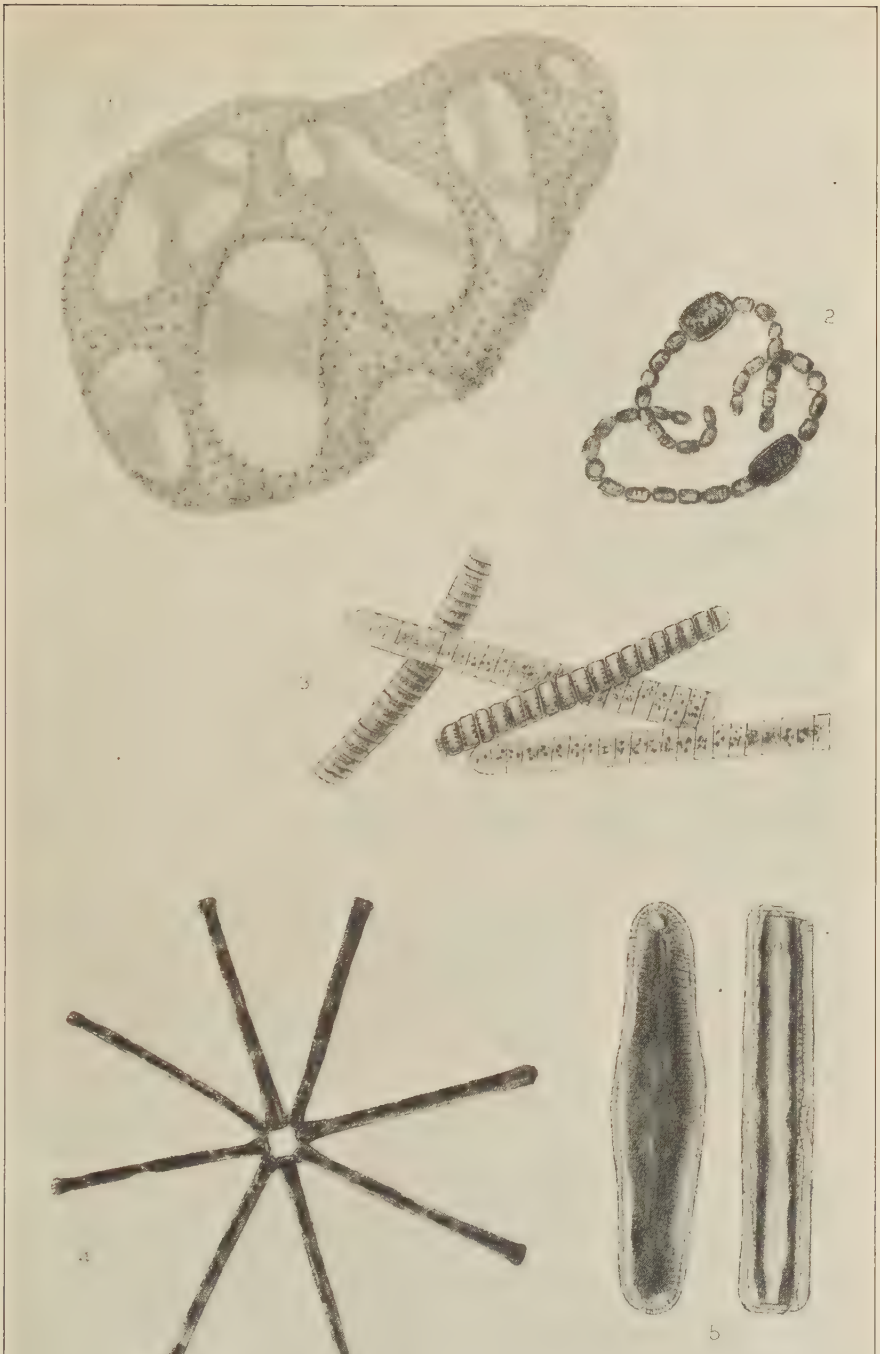
[1] The second edition can be obtained from the secretary of the association, 289 Fourth Avenue, New York City.

[2] In the methods for water analysis described in this book the standard methods have been closely followed, and due acknowledgment is here given to the splendid and self-effacing work of the committee that devised them.



ALGAE: 1, UROGLENA—X 300; 2, SPIROGYRA—X 500; 3, RESTING SPORES OF SPIROGYRA—X 500; 4, CHLAMYDOMONAS SHOWING RESTING CONDITION AND REPRODUCTIVE BODIES—X 1000.

FIG. 101.—ALGÆ COMMONLY FOUND IN WATER. [YEAR BOOK, U. S. DEP'T. OF AGR., 1902.]



ALGAE: 1, CLATHROCYSTIS – X 500; 2, ANABAENA – X 500;
3 OSCILLATORIA – X 500; 4, ASTERIONELLA – X 500;
5 NAVICULA SHOWING STRUCTURE OF DIATOM – X 500.

FIG. 102.—ALGÆ COMMONLY FOUND IN WATER. [YEAR BOOK, U. S. DEP'T. OF AGR., 1902.]

portant one, and explains how water may become "contaminated" by mere exposure to an impure atmosphere, as when an uncovered cistern is placed in a water-closet or when an overflow pipe is directly connected with a drain.

Method of Determining Odor.—The odor of the water should be observed both at room temperature and just below the boiling point. Odors may be detected at room temperature (20° C.) by shaking a sample violently in a gallon collecting bottle when it is half or two-thirds full; or by heating about 150 c. c. in a tall beaker without a lip and covered with a well-fitting watch glass. In either case care should be taken to observe the character of the odor the instant the receptacle is uncovered. The kind of odor observed may be described as vegetable, aromatic, grassy, fishy, earthy, moldy, musty, disagreeable, peaty, sweetish, etc., and the intensity by such terms as very faint, distinct, decided, or very strong.

The odors and tastes in water caused by microscopic organisms deserve special consideration, because they are common faults in water stored in open artificial reservoirs of all kinds. Certain organisms can be distinguished by their odor, as, for example, the "fishy" odor of *Uroglena,* which is a protozoon and classed with the *Infusoria;* the "aromatic" or "rose geranium" odor of *Asterionella,* which belongs to the *Diatomaceæ;* and the "pig-pen" odor of *Anabæna,* which is one of the blue-green algæ. These microscopic organisms mostly grow near the surface and require sunlight for their development; hence, odors produced by them never occur in covered reservoirs or in waters kept in the dark.

Calkins has shown that the odors caused by the undecomposed microscopic organisms are due to compounds of the nature of essential oils, and Whipple points out that the amount of such oil produced by an abundant growth of the organisms is quite sufficient to account for the effect observed. He notes for comparison that oil of peppermint can be recognized when diluted with water in the proportion of one part of oil to fifty million parts of water, and that when *Asterionella* is present to the extent of 50,000 organisms per c. c. the dilution of its oil is in the proportion of about one part to two million parts of water. Whipple further suggests that the flow of water through pipes may cause disintegration of organisms with liberation of the odor-producing oil, hence the odor at the tap may be greater than at the intake.

FIG. 103.—THE OIL DROPLETS IN A DIATOM.

The *Algæ* responsible for the vile tastes and odors in water do not depend upon organic matter or the bodies of other organisms for their

food supply. They require only carbonic acid and the nitrogen and mineral matters always present in the water and in the air, and the sunshine for their growth. In other words, they have properties comparable in many respects to the higher orders of chlorophyll-containing vegetation.

There are very many kinds of *algæ*, and they differ greatly in their odor-producing powers. Practically all American impounding reservoir waters suffer from them, but some far more than others. English reservoirs seem to be comparatively free from this nuisance, probably because of the lower temperatures of the surface waters. There is an average difference of at least 10° F. between the surface temperatures of English and American reservoirs.

A certain degree of quiet and repose is necessary for the development of a large growth of algæ; that is why they never develop to any extent in rivers and flowing water. Wave action from wind also prevents growth, and this seems to be the only reason why large lakes and reservoirs are less troubled by them than smaller ones.

In most American impounding reservoirs the water is drawn from near the surface layer, so as to avoid the odors and tastes of putrefaction in the bottom water, but it sometimes happens that the surface water is the more objectionable.

Prevention and Removal of Tastes and Odors.—The natural flow of water in the bed of a mountain stream over stones and ledges aerates it very well. This is nature's method of removing undesirable tastes and odors. Aeration may also be accomplished by bringing the water in contact with the air by devices such as fountains, waterfalls, etc. Such aeration always reduces, and sometimes removes, tastes and odors from the waters of reservoirs and small lakes, whether resulting from putrefaction in the stagnant bottom water or from growths of organisms in the surface water.

In general it may be stated that filtration alone is not efficient in removing tastes and odors; however, slow sand filtration has considerable power of reducing, and in some cases of removing, tastes and odors, but cannot be relied upon when the raw water is very bad.

Intermittent filtration is particularly successful in removing tastes and odors. It is successful because it brings the organic matter in contact with more air and in more intimate contact with air, and for a longer time in the pores of the sand, than can be secured in any other way.

It is practically impossible to prevent the seeding of reservoirs and ponds with algæ and other organisms responsible for the objectionable odors. The growth may be checked and the odors temporarily controlled by the use of copper sulphate (see page 800).

If a well becomes stagnant at the bottom, and thus develops vile

odors from putrefying organic matter, the trouble may be corrected by lowering the pump to near the bottom so as to prevent stagnation, or by filling up all unnecessary space with clean gravel and sand.

COLOR

Pure water, when viewed in small quantities, appears to be perfectly colorless, but, when viewed in bulk, as in the white-tiled baths at Buxton, and in certain Swiss lakes, it is seen to possess a beautiful greenish-blue tint. A very small amount of suspended or dissolved impurity is sufficient to obscure this color.

Impure waters almost invariably exhibit a color varying from green to yellow and brown, when examined through a depth of two feet in suitable tubes. It does not, however, follow that a colored water is, therefore, polluted or infected.

Color in surface water is usually of vegetable origin; animal matter contributes but little color. The coloring matter is extracted largely from dead leaves, bark, and roots, from soil, and from peat. It seems to be the same material as the coloring matter of tea, and it is certainly harmless, but it makes the water less pleasing in appearance, and great efforts have rightly been made to prevent it and to remove it. Water from swamps is usually highly colored, the degree of color depending upon the length of exposure.

Ground waters are usually colorless. If the water contains iron it will be perfectly clear on coming from the ground, but will soon turn a rusty yellow color. This is caused by the oxidation of the soluble ferrous salts to insoluble ferric salts.

Color in water should be distinguished from turbidity. True color is due to dissolved impurities, turbidity to substances in suspension. The "apparent color" is the color of the original sample, due to both dissolved and suspended matter.

The prevention of color in surface waters consists in draining swamps. Thus, in the catchment areas of the various reservoirs supplying Boston thousands of acres of swampy land have been drained for the purpose of reducing the color of the supplies, and with good results.

A colored water may be bleached by exposure to sunlight and air, but the bleaching of the water in reservoirs requires great storage capacity, and the drainage of swamps is likewise very expensive. Ozone applied in large amounts also destroys color, and the only objection to its use is the cost. Color may be removed to a considerable extent by simple filtration through sand. If the coloring matter is first rendered insoluble by the use of coagulants (sulphate of alumina), it is readily removed by filtration. Color is thus successfully removed from the

waters used by Norfolk, Va.; Charleston, S. C., and Watertown, N. Y. Sulphate of iron is less satisfactory as a coagulant than sulphate of alumina for the removal of color.

Method for Estimating Color.—Turbid waters should always be filtered before the color observations are made. The intensity of color may be determined by comparing with a standard platinum-cobalt solution; the tint or shade may be determined by comparison with the standard color disks of a Lovibond tintometer.

Platinum-Cobalt Standard.—The standard solution, which has a color of 500, is prepared as follows:

Dissolve 1.246 grams of potassium platinic chlorid ($PtCl_4$ 2KCl) containing 0.5 gram platinum, and one gram crystallized cobalt chlorid ($CoCl_2$ $6H_2O$) containing 0.25 gram of cobalt in water, with 100 c. c. concentrated hydrochloric acid, and make up to one liter with distilled water.

By diluting this solution with distilled water to the 100-c. c. graduation mark on the Nessler tubes, standards are prepared having colors of 0, 5, 10, 15, 20, 25, 30, 35, 40, 50, 60, and 70. These should be kept in Nessler tubes of such diameter that the 100-c. c. graduation mark is between 20 and 25 cm. above the bottom, and is uniform for all tubes. They should be protected from dust when not in use.

Procedure.—The color of a sample is observed by filling a standard Nessler tube to the graduation mark with the water to be examined, to a depth equal to that of the standards, and by comparing it with the standards. The observation should be made by looking vertically downward through the tubes upon a white surface placed at such an angle that light is reflected upward through the column of liquid.

Waters that have a color darker than 70 should be diluted before making the comparison, in order that no difficulties may be encountered in matching hues.

TURBIDITY

Practically turbidity is synonymous with muddiness. The turbidity of surface waters is usually due to clay or silt, also to finely divided organic matter, microscopic organisms, and a great variety of objects. Turbidity represents the amount of foreign substances in suspension; it is frequently, though incorrectly, spoken of as color. In a general way turbid waters exist in those regions where color is not found; the former represents the washings of a readily eroded drainage basin, the latter is mostly extracted from the decaying vegetation of swamps.

Pure water is clear and sparkling, in proportion to the amount of dissolved oxygen and carbonic acid. While brilliancy and clearness do not mean purity, on the other hand turbid waters are not necessarily

dangerous. A community for years may drink and seem satisfied with a turbid water that is little less than liquid mud. This was the case with Washington and the Potomac water, St. Louis and the Mississippi, and many other cities. When, however, such a city once appreciates the beautiful appearance of a clean water, they complain if the turbidity reaches the point of a faint opalescence. The turbidity question is practically limited to river waters. Ground waters should never be turbid, and, if so, should at once excite suspicion. Some ground waters become more or less turbid through the precipitation of iron.

All river waters are more or less turbid, but the differences are very great indeed. The amount of turbidity depends largely upon the character of the catchment areas. In general, rivers draining the large areas of our North and East, covered with glacial drift of a sandy character, are but little subject to turbidity. Thus, on an average, the Merrimac and Connecticut Rivers do not carry more than 10 parts per million of suspended matter. In that part of our country which is not glaciated, and this includes the lower Susquehanna basin, much of the Ohio basin, and the Missouri basin, and all to the south of them, turbidity is often present in large amounts, and consists largely of clay in extremely fine particles. The water often runs turbid in these streams continuously for weeks and even months at a time. The Missouri River carries the largest amount of sediment of any of our rivers largely used for water supply. The annual average runs as high as 1,200 or 1,500 parts of sandy matter per million. In winter it falls to 200 parts or less, while in midsummer it rises for weeks and even months to 5,000 parts or more.

If the turbidity is sufficiently coarse-grained it may be removed by sand filtration without previous chemical treatment. Very turbid waters can be cleared, in part, in settling basins; this lightens the work of the filters and reduces the cost. Scrubbers, which are preliminary rough filters, may also be used to protect the sand filters. In many instances the individual particles of clay which make up the turbidity are much smaller than the bacteria. They will not settle out, even after prolonged storage, and they cannot always be removed by filtration alone. There is only one known way of removing such turbidity, and that is by coagulation or chemical precipitation. The substances most commonly used for this purpose are: aluminium sulphate, alum, or sulphate of iron (see page 794).

With reference to the influence of the suspended matter upon health we find some conflict of opinion. Kober states that water containing 50 parts per 100,000 or 30 grains of solid matter per gallon is unfit for drinking purposes, on account of its irritating effects upon the gastrointestinal tract. Apart from this, turbidity appears to have no special sanitary significance.

Methods for Estimating Turbidity.—There are three methods by which the degree of turbidity may be determined: (1) the platinum wire method, which consists of determining the depth of water through which a platinum wire of standard diameter may be seen; (2) comparison with waters of standard turbidity, made by adding 1 gram of finely powdered diatomaceous earth to 1 liter of distilled water; this is known as the silica standard; and (3) the amount of suspended particles in water may be determined in special instruments known as turbidimeters or diaphanometers. These instruments consist of a graduated glass tube with a flat polished bottom, inclosed in a metal case. This is held over an English standard candle, and so arranged that one may look vertically down through the tube and see the image of the candle. The observation is made by pouring the sample of water into the tube until the image of the candle just disappears from view. The graduations on the tube correspond to turbidities produced in distilled water by certain numbers of parts per million of the silica standard.

The standard of turbidity adopted by the United States Geological Survey consists of a water which contains 100 parts of silica per million, in such a state of fineness that a bright platinum wire 1 millimeter in diameter can just be seen when the center of the wire is 100 millimeters below the surface of the water and the eye of the observer is 1.2 meters above the wire, the observations being made in the middle of the day in the open air, but not in sunlight, and in a vessel so large that the sides do not shut out the light so as to influence the results. The turbidity of such water is taken as 100, and all turbidity readings, by no matter what method used, should conform with this method.

REACTION

The alkaline reaction of natural waters ordinarily depends upon the carbonate and bicarbonate of calcium and magnesium. In some waters in the West it also includes the carbonate of sodium and of potassium. The alkalinity of water is determined by titrating 100 c. c. of the sample with $\frac{N}{50}$ sulphuric acid, using 0.5 c. c. of a solution of lacmoid as an indicator. The lacmoid solution consists of 2 grams in one liter of 50 per cent. alcohol. The last cubic centimeter or two of acid must be added while the sample is almost at the boiling temperature, and the end reaction is not read until a drop of acid, striking the surface of the liquid, sinks to the bottom of the dish without producing a change in the uniform reddish or purplish color of the solution. Erythrosin may be used as an indicator when it is desired not to use heat. The number of cubic centimeters of $\frac{N}{50}$ sulphuric acid used, when multiplied

by ten, gives the number of parts per million of alkalinity in terms of calcium carbonate.

Under certain circumstances rain water, water from peat bogs, and water from coal mines, tanneries, etc., have an acid reaction. In mining regions waters are frequently acid from high quantities not only of CO_2, but also of sulphuric acid and various sulphates—those of iron and aluminium giving an acid reaction. When these are present, the total acidity is determined by titrating the water in the cold with a standard sodium carbonate solution, using phenolphthalein as an indicator.

Mine water is that which is constantly flowing from the coal and surrounding strata. It is collected in ditches at one side of the gangways and tunnels, and is allowed to flow to the lowest point in the mine or to the foot of the shaft, from which it is pumped to the surface. Large quantities of this and other water are used to wash the coal. This water is acid, and it is now well known, from the researches of Dixon, Matson, and others in the anthracite coal regions of Pennsylvania, that such water has a destructive effect upon typhoid, colon, and other bacteria. The acidity of the streams in Pennsylvania is a large factor in neutralizing the pollution of the water supplies of Philadelphia, Pittsburgh, Harrisburg, and other cities. The spent tan liquors from tanneries are also acid, and are known to exert a somewhat similar influence on sewage organisms.

Rain water collected in the vicinity of towns has usually a slight acid reaction and acts upon lead. The free acid in rain water is apparently sulphuric, no doubt derived from the sulphur in the coal used.

Water from marshes, swamps, and especially from peat bogs may have a markedly acid reaction, especially in dry weather, when the flow will be comparatively small. Heavy storms wash out the water which has long been in contact with the decaying vegetation. The acidity in this case is due to organic acids.

When the collection of an acid water cannot be avoided, arrangements should be made for filtering through some material capable of completely neutralizing the acid, as without some such arrangement the consumers of the water run the risk of lead poisoning, provided lead service pipes are used. A river water suddenly turning acid in reaction plays havoc with a slow sand filter. This has occurred in the Pittsburgh filter.

TOTAL SOLIDS

The total solids or residue on evaporation is obtained by evaporating a given quantity of water to dryness, when a grayish-white residue, composed of mineral and some organic matter which has been held by

the water in suspension and in solution, will be obtained. The amount of this residue varies with the character of the water, and furnishes an index of the total quantity of foreign impurities, and further furnishes a rough index of the relative quantity of inorganic and organic substances which make up these impurities.

Method.—Place 100 c. c. of the water in a clean platinum dish. If the water is of high magnesium content add 25 c. c. of $\frac{N}{50}$ sodium carbonate solution to the water, and correct for this addition in the computation; evaporate to dryness on a water bath, and finish the evaporation for half an hour or to constant weight in a toluene oven at 103° C. Now place the platinum dish in a desiccator over sulphuric acid until cool, and weigh. The increase in weight gives the total solids, or residue on evaporation.

This residue is now heated to a dull red heat and the platinum dish again weighed. The difference in weight is called the "loss on ignition," and the weight of the substances remaining in the platinum dish is known as the "fixed residue." The loss on ignition is an index of the amount of organic matter in the water. A portion of the loss, however, may be due to ammonia or other volatile compounds and unstable mineral salts. The fixed residue is an index of the mineral content of the water. With waters low in organic matter, but relatively high in iron, the fixed residue is frequently used as a matter of convenience for the determination of iron. In water analysis it is usual to note the character of the odor upon ignition of the residue. This may be earthy, or may suggest organic matter of vegetable origin or animal origin.

The amount of total solids in a water depends upon the character of the soil with which the water has been in contact, the length of exposure, and the amount of carbon dioxid in the water to favor the solution of inorganic salts. Some mineral springs contain very large amounts of total solids, derived from deeply situated natural deposits, as, for example, the springs at Saratoga, Carlsbad, Kissingen, etc.

The permissible amount of solids as represented by the residue on evaporation, which consists of the dissolved mineral constituents, cannot be arbitrarily stated, but 500 parts per million are generally held as excessive.

HARDNESS

The quality of hardness in water is more of an economic question than one of sanitary interest, except, perhaps, as the encouragement of the use of soap and cleanliness is of fundamental importance in hygiene and sanitation. Hardness in water is due to the presence of the soluble salts of the alkaline earths—especially calcium and magnesium. These

salts form a curd with soap instead of a lather, hence more or less soap must be wasted in decomposing the lime and magnesia compounds before a lather will form. Thus, one grain of calcium carbonate, for example, will use up 8 grains of soap before a lather can be produced; in this way hard water causes an enormous waste of soap. In Europe, hardness is usually expressed in degrees. Each degree corresponds to one grain of carbonate of lime or its equivalent of other lime or magnesium salts in a gallon of water.

The hardness remaining after the water has been boiled is called "permanent hardness." The hardness that may be removed by boiling is known as "temporary hardness." This distinction between temporary and permanent hardness arises from the fact that calcium and magnesium carbonates are but slightly soluble in water. In the presence of carbonic acid the so-called bicarbonates are formed, which are much more soluble than the normal carbonates. One gallon of pure water will dissolve from 2 to 3 grains of the normal carbonates, but when the water contains carbonic acid it will dissolve 20 or more grains. Upon heating, the CO_2 is driven off, and the excess of normal carbonates of lime and magnesia is in consequence precipitated, thus reducing the hardness of the water.

Waters under 4 degrees of hardness may be considered soft, those exceeding 12 degrees hard. Fifty parts per million of calcium sulphate and chlorid of magnesium is usually regarded as excessive. Boiler scale is usually due to deposits of sulphates and carbonates of calcium and magnesium.

Rain water is always soft; surface waters vary, but are usually not very hard; ground waters are apt to be hard.

Two conditions must be present to make a ground water hard: first, the material through which the water passes must contain lime or magnesia, and, second, the conditions must be favorable for dissolving it. The latter practically means that CO_2 must be present.

Waters drawn from limestone regions vary greatly in hardness. Rain water contains but little carbonic acid and, therefore, has little power of dissolving lime. The principal source of the carbonic acid in ground water is from the soil, resulting from the decomposition of organic matter. The hardness of water, therefore, depends more upon the nature of the catchment area than upon the amount of lime in the various materials over which the water flows. Thus, the water supply of Vienna is comparatively soft, notwithstanding that it comes entirely from limestone rocks. The mountainous region which forms the catchment area is barren and sterile, and the water does not get the carbonic acid needed to dissolve the lime. The Winnipeg water drawn from limestone underlying the rich prairies is excessively hard. It is interesting to note that many deep well waters of eastern Massa-

chusetts are comparatively soft, although they contain large amounts of carbonic acid.

Very hard water may be softened upon a large scale with iron and lime. As a rule, methods of softening are required with ground waters rather than with surface waters.

The common method in use is the Clark process, in which lime is added to the water either in the form of freshly slaked lime or milk of lime. The calcium hydroxid unites with the carbon dioxid in the water, forming calcium carbonate, which is insoluble, and at the same time precipitates the calcium carbonate held in solution in the water by the CO_2. Sodium carbonate is used to reduce the permanent hardness of water due to sulphates.

Methods.—The hardness of water, both temporary and permanent, is determined by the soap method. By far the most accurate method of determining the true temporary hardness due to the bicarbonate alkalinity is to titrate the original sample of water, and also some of the water after boiling, with $\frac{N}{50}$ sulphuric acid, using lacmoid or erythrosin as an indicator, as already described under Reaction, page 731.

The soap method is carried out as follows: Measure 50 c. c. of the water into a 250-c. c. bottle and add the standard soap solution in small quantities at a time (from 0.2 to 0.3 c. c.), shaking the bottle vigorously after each addition until a lather forms over the entire surface of the water, and remains continuous for 5 minutes after the bottle is laid upon its side. From the amount of soap solution added, the quantity of calcium carbonate equivalent to each cubic centimeter of the soap solution is indicated in the following table:

TABLE OF HARDNESS, SHOWING THE PARTS PER MILLION OF CALCIUM CARBONATE ($CaCO_3$) FOR EACH TENTH OF A CUBIC CENTIMETER OF SOAP SOLUTION WHEN 50 C. C. OF THE SAMPLE ARE USED

c. c. of Soap Solution	0.0 c.c.	0.1 c.c.	0.2 c.c.	0.3 c.c.	0.4 c.c.	0.5 c.c.	0.6 c.c.	0.7 c.c.	0.8 c.c.	0.9 c.c.
0.0.....	....	....	....	....	....	....	....	0.0	1.6	3.2
1.0.....	4.8	6.3	7.9	9.5	11.1	12.7	14.3	15.6	16.9	18.2
2.0.....	19.5	20.8	22.1	23.4	24.7	26.0	27.3	28.6	29.9	31.2
3.0.....	32.5	33.8	35.1	36.4	37.7	38.0	40.3	41.6	42.9	44.3
4.0.....	45.7	47.1	48.6	50.0	51.4	52.9	54.3	55.7	57.1	58.6
5.0.....	60.0	61.4	62.9	64.3	65.7	67.1	68.6	70.0	71.4	72.9
6.0.....	74.3	75.7	77.1	78.6	80.0	81.4	82.9	84.3	85.7	87.1
7.0.....	88.6	90.0	91.4	92.9	94.3	95.7	97.1	98.6	100.0	101.5

In adding the soap solution to waters containing magnesium salts it is necessary to avoid mistaking the false or magnesium end-point for the true one. If the end-point was due to magnesium the lather now disappears. Soap solution must then be added until the true end-point

is reached. Usually the false lather persists for less than 5 minutes. Consequently, after the titration is apparently finished, read the burette and add about 0.5 c. c. of soap solution.

At best the soap method is not a precise test on account of the varying proportions of calcium and magnesium present in different waters. For the determination of hardness, especially in connection with processes for purification and softening, it is advisable to use volumetric or gravimetric methods.

ORGANIC MATTER

It is not possible to determine the amount of organic matter present in a sample of water by any direct method. As all proteid matter contains nitrogen, methods have been devised to determine the total amount of nitrogen and also the amount of nitrogen in various combinations. From such data valuable information concerning the sanitary history and sanitary quality of the water may be inferred. The nitrogen is determined as (1) total nitrogen; (2) nitrogen as free ammonia; (3) nitrogen as albuminoid ammonia; (4) nitrogen as nitrites; (5) nitrogen as nitrates.

The organic matter in water is of animal and vegetable origin and exists both in solution and in suspension. Some of it is in the body of living beings; some of it is in their dead bodies; and some of it is in various stages of decomposition until the final stable compounds, such as ammonia and nitrates, are reached. The total amount of organic matter present in a sample of water is represented by the amount of nitrogen as free ammonia and albuminoid ammonia. The presence of nitrogen as nitrites and nitrates indicates the amount of self-purification which the water has undergone. Their significance will be discussed separately.

Free Ammonia.—If there is much free ammonia in the water the sample may be nesslerized directly. If the water contains comparatively little, as is usually the case, the ammonia must first be concentrated by distillation and condensation.

Place 500 c. c. of the sample of water in a metal or glass still connected to a tin or aluminium condenser in such a way that the distillate may be conveniently delivered directly into Nessler tubes. The entire apparatus must first be freed from ammonia by blowing steam through it until the distillate shows no trace of free ammonia. When this has been done the distilling flask is emptied and 500 c. c. of the sample of water measured into it. The distillation should be carried on at a rate so that not more than 10 c. c. nor less than 6 c. c. condense per minute; that is, it should take from 5 to 10 minutes to distill 50 c. c., which is the quantity Nessler tubes are ordinarily graduated to

contain. Three Nessler tubes of the distillate containing 50 c. c. each are collected from the first portion that comes over; these contain the free ammonia.

If the sample is acid, or if the presence of urea is suspected, about one-half gram of sodium carbonate should be added previous to distillation, otherwise the ammonia will not come off. Sodium carbonate is omitted, when possible, as it tends to increase "bumping."

The amount of ammonia is determined by adding 2 c. c. of Nessler reagent to each tube and comparing the depth of color with a set of standard tubes prepared with a known quantity of ammonium chlorid solution, plus an equal quantity of Nessler reagent.

Nessler's reagent is prepared by dissolving 50 grams of potassium iodid in a minimum quantity of cold water. To this add a saturated solution of mercuric chlorid until a slight permanent precipitate persists. Then add 125 grams of potassium hydroxid dissolved in 250 c. c. of water, allowing it to clarify by sedimentation before using; dilute to one liter, allow to stand, and decant. The solution should give the required color with ammonia within 5 minutes after addition, and should not precipitate with small amounts of ammonia within 2 hours. The reaction between Nessler's reagent and ammonia is an empyric one. The HgI_2 2KI KOH, which constitutes the Nessler's reagent in the presence of ammonia, forms a brownish compound, known as mercurammonium iodid, and having the formula $NHg_2I\ H_2O$.

Standard NH_4Cl Solution.—The standards for comparison consist of ammonium chlorid dissolved in ammonia-free water. Dissolve 3.82 grams of ammonium chlorid in 1 liter of water; dilute 10 c. c. of this to 1 liter with the ammonia-free water. One c. c. will then equal 0.00001 gram of nitrogen.

A gram molecule of NH_4Cl weighs 53.5 grams—that is:

$$N\ 14+H\ 4+Cl\ 35.5=53.5$$

The equation would then be:

$$14:53.5::1:x$$
$$x=3.82$$

That is, if there are 14 grams of nitrogen in 53.5 grams of ammonium chlorid, then 1 gram of nitrogen is contained in 3.82 grams of ammonium chlorid. It is to be noted that, while the method determines the amount of ammonia, the results are expressed in terms of nitrogen. In the same way the nitrites and nitrates are also expressed in terms of nitrogen.

Prepare a series of 16 Nessler tubes, which contain the following number of cubic centimeters of the standard ammonium chlorid solution, namely: 0.0, 0.1, 0.3, 0.5, 0.7, 1.0, 1.4, 1.7, 2.0, 2.5, 3.0, 3.5, 4.0,

4.5, 5.0, 6.0; dilute each one with 50 c. c. of the standard ammonia-free water. These will contain 0.00001 gram of nitrogen for each cubic centimeter of the standard solution used. Add 2 c. c. of the Nessler reagent to each tube; do not stir the contents of the tubes.

The color produced in the distillate from the sample under examination is now compared with standards by looking vertically downward through them at a white surface placed at an angle in front of a window so as to reflect the light upward. The tubes should be allowed to stand at least 10 minutes after nesslerizing before making the comparison.

The last 50 cubic centimeters of the distillate examined should contain no ammonia, or at most a trace, otherwise it may be inferred that all has not been collected, or some error has crept into the work. It is not uncommon for the last tube to contain a little ammonia when the organic matter is of plant origin. Ammonia determinations should be carried out in a special room, where at least volatile ammonia reagents are not exposed. Special care must be exercised not to contaminate the Nessler tubes with soiled fingers, rags, etc. Care must be exercised to thoroughly wash the tubes free from alkaline soaps. The Nessler tubes containing the standard solution and the samples for comparison should be at the same temperature, and other conditions should be as alike as possible.

Example.—

The first Nessler tube=2.5 c. c. standard NH_4Cl solution, or 0.000,025 gram N as NH_3.

The second Nessler tube=0.7 c. c. standard NH_4Cl solution, or 0.000,007 gram N as NH_3.

The third Nessler tube=0.0 c. c. standard NH_4Cl solution, or 0.000,000 gram N as NH_3.

Total, 0.000,032 gram N as NH_3.

(Note: 1 c. c. of the standard solution contains 0.00001 gram of N as NH_3.)

Only 500 c. c. of the sample of water was distilled. We must, therefore, multiply by 2 in order to obtain the amount of N in one liter:

$$0.000,032 \times 2 = 0.000,064 \text{ gram of N as } NH_3 \text{ per } 1,000 \text{ c. c.}$$

If 1,000 c. c. contains 0.000,064 gram of N as NH_3, 1,000,000 parts will contain 0.064 part of N as NH_3—usually expressed as 0.064 part per million.

A simpler method of making the calculation is as follows:

$$2.5 + 0.7 \times 0.02 = 0.064 \text{ part per million.}$$

Significance of Free Ammonia.—The free ammonia which comes

off with the first part of the distillate usually exists in the water as chlorids or carbonates. It is called "free ammonia" because these salts are readily decomposed and the ammonia is expelled by boiling.

Rain water washes down some free ammonia which is found in the atmosphere. Angus Smith and Boussingault place the average amount of ammonia in the rain of temperate climates as 0.5 part per million.

The amount of ammonia in rain water was studied by Filhol. He found that in the city of Toulouse the rain water contained 6.60 parts per million, while the rain water collected near the city contained only from 0.44 to 0.77 part per million. These figures show the marked difference between city and country rain.

In a surface or ground water free ammonia represents one of the latter stages of putrefaction of organic matter; thus, the bacterial decomposition of sewage yields ammonia in abundance.

The ammonia itself ordinarily found in drinking water is harmless; its significance lies in the fact that it indicates the presence of putrefying organic matter.

The presence of free ammonia in clean and properly stored rain water has much less significance than in a surface or ground water.

Free ammonia in water results not only from the decomposition of nitrogenous organic matter, but is also formed during the process of denitrification, by which nitrates are again reduced to nitrites and nitrites to ammonia. This action only takes place near the surface of the soil, and to a limited extent. Deep well waters of exceptional purity upon chemical analysis, and practically sterile upon bacteriological examination, may contain a relatively high percentage of free ammonia. This is supposed to come from a chemical reduction under high pressure and perhaps temperature of the geological nitrogenous matter in coal and alluvial deposits.

A definite permissible limit for the amount of free ammonia which good water should contain cannot be fixed. Its significance must be judged from the other constituents of the water and a sanitary survey of its source. As a rule, pure water may contain from 0.015 to 0.03 or even 0.055 part per million. In general, free ammonia is less of a danger signal than the fixed or albuminoid ammonia.

Albuminoid Ammonia.—Nitrogen as albuminoid ammonia is always determined in conjunction with and as a continuation of the method for determining nitrogen as free ammonia. After obtaining 150 c. c. (that is, 3 Nessler tubes of 50 c. c. each) from the first portion of the distillate, for the purpose of determining nitrogen as free ammonia, withdraw the flame and add 40 c. c. or more of alkaline potassium permanganate, and continue the process until at least 4 portions of 50 c. c. each, or, preferably, 5 portions, of the distillate have been collected in separate Nessler tubes.

The alkaline potassium permanganate solution is made by pouring 1,200 c. c. of distilled water into a porcelain dish holding 2,500 c. c.; boil 10 minutes and turn off the gas. Add 16 grams of C. P. potassium permanganate and stir until dissolved. Then add 800 c. c. of 50 per cent. clarified solution of potassium or sodium hydrate and enough distilled water to fill the dish. Boil down to 2,000 c. c. Test each batch of this solution for albuminoid ammonia by making a blank determination. Correction should be made accordingly.

After the readily decomposed ammonia salts have been broken up and the ammonia driven off in the steam which condenses to form the first 150 c. c., the remainder of the sample of water in the still contains nitrogenous organic matter that requires a strong oxidizing agent to disintegrate it. This is accomplished by the alkaline potassium permanganate. The nitrogen in the complex protein molecule finally forms ammonia, and hence this is called albuminoid ammonia; the amount of it is determined by nesslerization, precisely as for free ammonia. In ground waters and surface waters containing but little pollution the nitrogen as albuminoid ammonia usually approximates about one-half of the total organic nitrogen. In sewage and other liquids containing considerable nitrogenous organic matter the percentage of ammonia forming organic matter is variable. For this reason the amount of albuminoid ammonia obtained by the alkaline permanganate method is less valuable than the total organic nitrogen determined by the Kjeldahl method.

If it is desired to determine how much of the organic matter is in solution and how much in suspension, the sample of water should be passed through a Berkefeld filter. The albuminoid ammonia in the filtrate represents the dissolved organic matter, and the difference between the albuminoid ammonia in the total sample and the filtered sample gives the suspended nitrogen as albuminoid ammonia.

The albuminoid ammonia is a fairly correct index of the amount of organic pollution in the water. It comes from minute organisms, both living and dead, that are in the sample, also from particles of animal and vegetable matter in suspension, and finally from the nitrogenous substances in solution and in various stages of decomposition. The organic matter in itself is not dangerous to health, but is undesirable because it putrefies and thus gives a water disagreeable tastes and odors; further, it offers food for bacterial growth. The amount of albuminoid ammonia is therefore an index of pollution, but if of vegetable origin it has much less sanitary significance than if of animal origin. Organic matter of animal origin yields a much larger amount of albuminoid ammonia than a similar amount of vegetable matter. Whether the organic matter comes from sewage, from a dead carcass, or from the swamps, cannot be stated with certainty from this test, but

if the albuminoid ammonia comes over quickly, that is, if most of it appears in the first Nessler tube, it is presumably of animal origin; whereas, if the ammonia comes over more slowly and the second and third Nessler tubes contain appreciable amounts, the organic matter is presumably of vegetable origin.

No arbitrary standard can be set as to the maximum amount of albuminoid ammonia a good water may contain. Waters considered "pure" often contain as much as 0.079 to 0.34 part of nitrogen as albuminoid ammonia per million.

Nitrites.—Nitrites in water are regarded as a special danger signal. The reason for this is that nitrites indicate that active putrefaction of nitrogenous organic matter is going on as the result of bacterial activity. The presence of nitrites, therefore, at once suggests organic pollution. The presence of nitrites in water represents the transitional stage in the oxidation of organic matter between ammonia and nitrates, and therefore indicates incomplete oxidation of the protein and the active growth of bacteria.

Nitrites are never present except in small amounts, for they are soon oxidized to the higher and more stable nitrates, but the minutest trace, according to some authorities, is sufficient to condemn a water. As a rule, pure water contains no nitrites, or traces only; on the other hand, nitrites may be absent from an impure water, owing to the fact that the oxidation has not reached this stage, or perhaps has entirely passed it. The absence of nitrites, therefore, does not mean that the water is necessarily safe, while their presence in any but the smallest measurable amounts shows pollution. We must not give to the nitrites an exaggerated importance: they are a danger signal in the same sense that the colon bacillus is a danger signal, indicating pollution but not necessarily infection, for they do not tell the source or nature of the organic matter. It should be remembered that the colorimetric test for nitrites with sulphanilic acid and α-amidonaphthylamin is one of the most delicate tests in chemistry. With this method we are able to detect quantities as small as one part in a hundred million. When, therefore, a water analyst reports a trace of nitrites it means an exceedingly minute quantity.

Nitrites are not only formed by the nitrifying bacteria in the soil from ammonia, but are also formed from the denitrification of nitrates by a variety of microorganisms. The typhoid bacillus, the colon bacillus, and many other bacteria have the power of producing nitrites in culture media.

Nitrites are poisonous, but the minute amounts found in water can scarcely have a pharmacological effect.

Method for Estimating Nitrogen as Nitrites.—*Reagents:* (1) Sulphanilic acid solution. Dissolve eight grams of the purest sul-

phanilic acid in 1,000 c. c. of 5 N. acetic acid (sp. gr. 1.041). This is practically a saturated solution.

(2) α-amidonaphthalene acetate solution. Dissolve 5.0 grams solid α-naphthylamine in 1,000 c. c. of 5 N. acetic acid; filter the solution through washed absorbent cotton.

(3) Sodium nitrite, stock solution. Dissolve 1.1 grams silver nitrite in nitrite-free water; precipitate the silver with sodium chlorid solution and dilute the whole to one liter.

(4) Standard sodium nitrite solution. Dilute 100 c. c. of solution (3) to one liter; then dilute 10 c. c. of this solution to one liter with sterilized nitrite-free water; add one c. c. of chloroform and preserve in a sterilized bottle. One c. c. = 0.000,000,1 gram nitrogen.

Procedure.—Measure out 100 c. c. of the decolorized sample (decolorized by adding aluminium hydrate free of nitrite—see under Chlorin), or a smaller portion diluted to 100 c. c., into a Nessler tube. At the same time make a set of standards by diluting various volumes of the standard nitrite solution in Nessler tubes to 100 c. c. with nitrite-free water, for example, 0, 1, 3, 5, 7, 10, 14, 17, 20, and 25 c. c. Add 2 c. c. of reagents Nos. 1 and 2 (above) to each 100 c. c. of the sample and to each standard. Mix; allow to stand 10 minutes. Compare the samples with the standards. Do not allow the samples to stand over one-half hour before being compared, on account of absorption of nitrites from the air. Make a blank determination in all cases to correct for the presence of nitrites in the air, the water and other reagents. Dilute all samples which develop more color than the 25 c. c. standard before comparing. Mixing is important.

When 100 c. c. of the sample are used, then 0.001 times the number of c. c. of the standard gives the parts per million of nitrogen as nitrite.

Nitrates.—Nitrates are the end products of the mineralization of organic matter. Their presence, therefore, signifies past or distant pollution. While the absence of nitrates does not necessarily mean purity, their presence, on the other hand, does not necessarily indicate immediate danger. If a water contains an appreciable quantity of nitrates and no nitrites, it shows that the source of pollution has been distant and that the organic matter has been completely oxidized. In waters considered pure the nitrates are rarely less than 0.3 part, or they may run as high as 1.6 parts, per million. Impure waters may contain very much more, as 17, 20, or more parts per million. Nitrates usually exist in water as salts of alkaline bases. The test for nitrates depends upon the fact that they react with phenoldisulphonic acid to form a compound resembling picric acid, which is yellow in the presence of an alkali. The amount of nitrates is determined colorimetrically by comparison with standard solutions.

Phenolsulphonic Acid Method for Nitrates.—*Reagents:* (1)

Phenolsulphonic acid. Mix 30 grams of synthetic phenol with 370 grams of C. P. concentrated sulphuric acid in a round-bottom flask. Put this flask in a water bath and support it in such a way that it shall be completely immersed in the water. Heat for six hours.

(2) Ammonium hydrate solution diluted with distilled water, 1 to 1. Potassium hydrate may be used. The ammonia gives a better color than the potassium, but should not be used if this test is carried on in the same room where free and albuminoid ammonia are being determined for fear of false results from contamination.

(3) Standard nitrate solution. Dissolve 0.72 gram of pure recrystallized potassium nitrate in one liter of distilled water. Evaporate cautiously 10 c. c. of this strong solution on the water bath. Moisten quickly and thoroughly with 2 c. c. of phenolsulphonic acid and dilute to one liter for the standard solution; one c. c. of which equals .000,001 gram of nitrogen.

Procedure.—Evaporate 20 c. c. or less of the sample of water in a small porcelain evaporating dish on the water bath, removing it from the bath just before it has come to dryness. Let the last few drops evaporate at room temperature in a place protected from the dust. When the sample is suspected to contain a large amount of nitrate, evaporate less than 20 c. c. If it is suspected to contain but little, evaporate more.

If the sample has a high color, decolorize before evaporating by the use of washed aluminium hydrate, as directed in connection with the chlorin determination.

Add 1 c. c. of phenolsulphonic acid and rub this quickly and thoroughly over the residue with a glass rod. Add about 10 c. c. of distilled water and stir with a glass rod until mixed. Add enough ammonium hydrate solution (or potassium hydrate if the operation must of necessity be carried on in a room where ammonia distillations are made) to render the liquid alkaline. Transfer the liquid to a 100 c. c. Nessler tube and fill the tube to the 100 c. c. mark with distilled water.

If nitrates are present there will be formed a yellow color; this may be compared with permanent standards made for the purpose, which keep satisfactorily for several weeks. The series of standards for comparison shall be made by putting varying quantities of the standard solution into 100 c. c. tubes and making up to the 100 c. c. mark with distilled water, adding 5 c. c. of strong ammonia to each tube, in accordance with the table on page 744.

Compare the sample treated as above described with these standards by looking down vertically through the tubes at a white surface so placed in front of a window that it will reflect the light upward through them.

If the figures obtained by this comparison in cubic centimeters of standard added be divided by the number of c. c. of the sample which

were evaporated, the quotient gives the number of parts per million of nitrogen in the form of nitrate.

TABLE

Amount of Dilute Standard Added	Standard Nitrate	Amount of Dilute Standard Added	Standard Nitrate
c. c.	milligram	c. c.	milligram
0.0	0.000	15.0	0.015
1.0	0.001	20.0	0.020
3.0	0.003	25.0	1.025
5.0	0.005	30.0	0.030
7.0	0.007	35.0	0.035
10.0	0.010	40.0	0.040

CHLORIN

Chlorin as sodium chlorid or common salt is a normal constituent of all waters. Traces of it are found in rain water taken up from the air, especially near the sea-coast. The rain water collected at Troy, New York, was found by Mason to average 1.64 parts per million of chlorin. The amounts varied from 0.75 part per million in April to 3 parts per million in October. The chlorin in surface and ground waters, generally speaking, comes from the mineral deposits in the earth: from the ocean vapors and spray carried inland by the wind; also from polluting materials like sewage and trade wastes, both of which are apt to contain the common salt used in the household and in manufacturing. A comparison of the chlorin content of a water with that of other waters in the general vicinity known to be unpolluted frequently affords useful information as to its sanitary quality.

Before the water analyst is able to properly interpret the significance of the chlorin content of a water it is necessary to know the normal amount of chlorin present in the waters of that locality. Thus, surface waters near Provincetown, on Cape Cod, contain from 23 to 24 parts of chlorin per million, while surface waters near Boston contain from 3 to 6 parts per million. Near the middle of the state of Massachusetts (Worcester) the surface waters contain only 1.2 to 1.9 parts per million, while in the western portion of the state, farthest from the sea, the surface waters contain but 0.7 to 0.9 parts per million. The amount of normal chlorin in the waters of Massachusetts has been carefully studied by the State Board of Health, and a map has been issued showing the isochlors, or normal chlorine lines.

In Massachusetts the whole of the surface of the country, with the exception of a very small portion, is non-calcareous, and the surface waters carry but little chlorin in composition, if unpolluted, the amount

of chlorin decreasing continuously from the coast inland. In a report on the State water supplies, 1887-1890, the Commissioners state that "in a general way 4 families or 20 persons per square mile will add, on an average, .01 of a part per 100,000 of chlorin (.1 part per million) to the water flowing from this area, and that a much smaller population will have the same effect during seasons of low flow."

The amount of chlorin in a water of a district varies with several factors, such as the distance from the sea, the amount of rainfall, the amount of evaporation, and the direction of the winds; an increase over the normal is an indication of pollution, and comes mostly from urine. While the ammonia and the nitrites may have disappeared and the nitrates may have been largely taken up by growing vegetation, the chlorin salts, which are exceedingly stable, will be left to indicate remote or passed pollution.

The mixture of even a small proportion of sea-water renders the water hard and salty and undesirable for domestic use. Magnesium chlorid also renders a water unsuitable for use in boilers. Wells driven near the sea frequently become mixed with sea-water, particularly if sufficient water is withdrawn to cause suction. When this happens the sea-water passes back under the wells as an undercurrent and gradually mixes with the fresh water above it and sooner or later appears in the well. When this happens it may be a slow and hard process to operate the well so as to avoid drawing sea-water. In wells near the sea it is important to draw no more fresh water than would otherwise flow to the ocean. This is often a difficult problem to arrange so as to get the maximum quantity of water obtainable. This sea-water question has been more thoroughly and scientifically studied in Holland than elsewhere.

Determination of Chlorin.—*Reagents.* (1) Standard salt solution. Dissolve 16.48 grams of fused sodium chlorid in one liter of distilled water. Dilute 100 c. c. of this stock solution to one liter in order to obtain a standard solution, each c. c. of which contains .001 gram of chlorin.

(2) Silver nitrate solution. Dissolve about 2.40 grams of silver nitrate crystals in one liter of distilled water. One c. c. of this will approximately equal .0005 gram of chlorin. Standardize this against the standard salt solution.

(3) Potassium chromate. Dissolve 50 grams of neutral potassium chromate in a little distilled water. Add enough silver nitrate to produce a slight red precipitate. Filter and make up the filtrate to one liter with distilled water.

(4) Aluminium hydrate. Dissolve 125 grams of potash or ammonium alum in one liter of distilled water. Precipitate the aluminium hydrate by cautiously adding ammonium hydrate. Wash the precipitate

in a large jar by the successive addition of distilled water and by decantation until free from chlorin, nitrites, and ammonia.

Procedure.—For this determination where the chlorin content is not extremely low or very high, titrate 50 c. c. of the sample in a white six-inch porcelain evaporating dish with the standard silver nitrate solution. If the chlorin is very high in amount, use 25 c. c., or even a smaller quantity if desired, diluting the volume taken with distilled water to 50 c. c. When the sample is very low in its chlorin content, more accurate results may be obtained by using 50 c. c. of the sample and adding, prior to titration, one c. c. of standard salt solution.

OXYGEN

Oxygen Consumed.—The oxygen consumed means the oxygen which the organic compounds in water consume when treated in an acid solution with potassium permanganate. The expression is synonymous with "oxygen required" or "oxygen absorbed." Oxygen consumed is, therefore, an index of the amount of putrescible organic matter present and should carefully be distinguished from the expression "dissolved oxygen," which refers simply to the amount of oxygen held in solution by the water.

It is the carbon and not the nitrogen in organic matter which is oxidized by potassium permanganate in an acid solution; hence this determination is frequently referred to as an indication of the carbonaceous organic matter present. The method indicates only a certain portion of the carbon, and this ratio varies in different samples of water. Further, it does not differentiate the carbon present in unstable organic matter from that in what might be called fairly stable organic matter, such as is sometimes referred to as "residual humus." The presence of nitrites, ferrous iron, sulphids, or other unoxidized mineral compounds causes oxygen to be taken up and hence increases the amount of oxygen consumed by this method. In case such substances are present, a correction should be made when studying carbonaceous organic matter.

Determination of Oxygen Consumed.—*Reagents.* (1) Dilute sulphuric acid. One part of sulphuric acid to three parts of distilled water. This shall be freed from oxidizable matters by adding potassium permanganate until a faint pink color persists after standing several hours.

(2) Standard potassium permanganate solution. Dissolve 0.4 gram of the crystalline compound in one liter of distilled water. Standardize against an ammonium oxalate solution. One c. c. is equivalent to 0.0001 gram of available oxygen.

(3) Ammonium oxalate solution. Dissolve 0.888 gram of the sub-

stance in one liter of distilled water. One c. c. is equivalent to 0.0001 gram of oxygen.

(4) Potassium iodid solution. Ten per cent. solution free of iodate.

(5) Sodium thiosulphate solution. Dissolve 1.0 gram of the pure crystallized salt in one liter of distilled water. Standardize against a potassium permanganate solution which has been standardized against an ammonium oxalate solution. As this solution does not keep well, determine its actual strength at frequent intervals.

(6) Starch solution. Mix a small amount of clean starch with cold water until it becomes a thin paste; stir this into 150 to 200 times its weight of boiling water. Boil for a few minutes, then sterilize. It may be preserved by adding a few drops of chloroform.

Procedure.—Measure into a flask 100 c. c. of the water, or a smaller diluted portion if the water is of high organic content. Add 10 c. c. of sulphuric acid solution and 10 c. c. of potassium permanganate solution, and allow the treated sample of water to digest 30 minutes at boiling temperature in a water bath.

Precisely at the end of the period of digestion remove the flask and add 10 c. c. of the ammonium oxalate solution. Titrate with the permanganate solution until a faint but distinct color is obtained.

Each c. c. of the permanganate solution in excess of the oxalate solution represents 0.0001 gram of oxygen consumed by the sample.

At the end of the period of digestion, if not made at the boiling temperature, add 0.5 c. c. of potassium iodid solution to discharge the pink color; mix; titrate the liberated iodin with thiosulphate until the yellow color is nearly destroyed; then add a few drops of starch solution and continue titration until the blue color is just discharged.

Should the volume of permanganate solution be insufficient for complete oxidation, repeat the analysis, using a larger volume, so that at least three c. c. of the permanganate solution will be present in excess when the ammonium oxalate solution is added.

When unoxidized mineral substances, such as ferrous sulphate, sulphids, nitrites, etc., are present in the sample, corrections should be applied as accurately as possible by procedures suitable for the samples being analyzed. Direct titration of the acidified sample in the cold, using a three-minute period of digestion, serves this purpose quite well for polluted surface waters and fairly well for purified sewage effluents. Raw sewages containing no trade wastes seldom need such a correction, but when raw sewages contain "pickling liquors" it is important. In all samples containing both unoxidized mineral compounds and gaseous organic substances the latter should be driven off by heat and the sample allowed to cool before applying this test for the correction factor. Where such corrections are necessary the fact should be stated, with the amount of correction.

This is one of the oldest methods for determining organic matter and has been in very wide use for more than half a century. It was introduced as soon as the fact was recognized that the loss on ignition of the residue upon evaporation may indicate certain volatile mineral matters, as well as organic matter. To-day the method of determination of oxygen consumed is ordinarily not included in a water analysis for the reason that the results vary widely, depending on the procedure as to certain details of the method, and from the further fact that the determinations of the organic matter in water may be more conveniently and satisfactorily estimated from the free and albuminoid ammonia.

Dissolved Oxygen.—Dissolved oxygen is another expression for the degree of aeration or oxygenation of water. It varies from zero to saturation or slight supersaturation. The amount of oxygen in solution is fairly constant in waters of uniform composition freely exposed to the air. Water containing sewage and other oxidizable matters uses up the dissolved oxygen. In badly polluted streams so much of the dissolved oxygen may be lost in this way that fish cannot breathe. They die from suffocation rather than from the toxic effects of the sewage. Water may contain practically no oxygen at depths of 40 or 50 feet, but deep soundings show that aeration probably exists to greater depths, for fish and aerobic organisms live at the bottom of the sea. In this case the oxygen may possibly be obtained from sources other than the dissolved oxygen from the air.

Dissolved oxygen makes water sparkling and palatable and also helps to consume the organic matter. Its absence permits the growth of anaerobic organisms that cause putrefaction and impart putrid tastes and odors to the water. Pasteur's original conception of fermentation was decomposition in the absence of oxygen.

The amount of oxygen found in the water of a running stream taken at different points may furnish valuable information as to the rapidity with which the process of self-purification is taking place from a chemical standpoint.

The amount of oxygen dissolved in a water may be measured by three methods: viz., that of Winkler, Thresh, or Levy. The method of Winkler is generally used in this country and possesses the advantage of requiring only simple and not readily breakable apparatus. It is therefore recommended as the standard method.

Method.—To determine the amount of dissolved oxygen it is necessary to collect the sample with extreme care in order to avoid the entrainment of any oxygen from the atmosphere. The sample bottles should be glass-stoppered, with a narrow neck, holding at least 250 c. c. The exact capacity of the bottle must be determined. The bottle should be filled through a glass or rubber tube which reaches to the bottom of the bottle, and the water allowed to overflow for several minutes, after

which the glass stopper is carefully replaced, so that no bubble of air is caught beneath it.

The method depends upon the fact that manganous sulphate in alkaline solution is oxidized to a manganate in the presence of oxygen in solution in water. On neutralization with sulphuric acid the manganese tends to revert to the manganous sulphate with the liberation of oxygen, and if potassium iodid is present this is decomposed by the liberated oxygen, setting free iodin. The liberated iodin is titrated with sodium thiosulphate, the end point being made more definite by the use of starch paste, which is added near the end of the titration.

IRON

Iron in water influences its quality from the standpoint of desirability rather than from the standpoint of health. After hardness there is no question of greater practical importance in considering the quality of a water. All natural waters contain a certain amount of iron, and ground waters are apt to contain it in objectionable amounts. Appreciable amounts of iron render water unsuitable for domestic and technical purposes; it stains clothes in the laundry, and is apt to cause headache and constipation if used habitually for drinking.

When iron is present in water it supports a fungus (*Crenothrix kuehniana*), an organism which may grow in the pipes in sufficient amount to obstruct the flow of water or even completely choke the pipe. It is chiefly troublesome in ground waters containing organic matter and iron. This was the cause of the complete obstruction of the water pipes in the New York Custom House in 1907. The same sometimes occurs in the pipes of driven wells.

Iron is very widely distributed and exists in practically all sands, gravels, soils, and rocks with which water comes in contact. The solution of the iron is brought about by the organic matter. The iron exists in the soil as ferric compounds. These are reduced by the organic matter to ferrous salts, which are soluble in water containing carbonic acid. Trouble from iron is always to be expected when there is an excess of organic matter in the material through which the water passes. In a well-drained, pervious soil the oxygen from the air circulates in the pores of the soil and furnishes what is required for the oxidation of the organic matter. Iron is not dissolved under these conditions, even in the presence of large amounts of organic matter, but if the air supply is cut off, as for instance in case the pores of the soil are filled with water, the solution of iron is sure to take place. The iron is dissolved in the form of ferrous salts, usually ferrous carbonate. When ground waters containing iron are first drawn they look clear, but the ferrous

salts in solution are soon oxidized on contact with the air to insoluble ferric salts, which are precipitated as red oxids.

Iron Pipes.—Nearly all waters attack iron pipes, corroding them and forming tubercles on the inner surface. This is objectionable, because it reduces the carrying capacity of the pipe and also influences the quality of the water.

Tubercles are formed as follows: The organic matter in the water settles in the pipe and attacks the iron through a blow hole or other minute opening in the coating. The organic matter decomposes, forming carbon dioxid, which acts upon the iron, causing some of it to go into solution as ferrous carbonate. The soluble ferrous carbonate for the most part passes on in the flowing water, but some of it becomes oxidized by the oxygen in the flowing water and is precipitated as the insoluble ferric carbonate and remains at the surface of the deposit. The iron precipitated in this way acts as a coagulant upon the organic matter in the flowing water at the point where the iron is precipitated. It thus attracts the organic matter from the flowing water and binds it to that previously deposited into a firm, compact, but porous mass, and this mass is the beginning of a tubercle. The process is continuous, though slow. Many years may elapse before the tubercle reaches the height of an inch. Tuberculation starts more freely and progresses more rapidly in waters from rivers or reservoirs containing suspended organic matter. It is less troublesome with filtered waters, and with lake waters relatively free from such suspended matter. Tuberculation may be prevented by improving the quality of the water or by thoroughly coating the inside of the pipes with asphaltum or tar. Cement-lined pipes are not subject to tuberculation, but have defects in other particulars. When the process has advanced far it may be corrected by pipe scrapers. They consist of appliances driven by the water pressure through the pipes, with arrangements to scrape off the tubercles. This temporarily restores the original carrying capacity of the pipe, but the process must be repeated at intervals. It has the disadvantage of also scraping off a large part of the tar coating and leaving the iron of the pipe exposed to the action of water to a much greater extent. (Hazen.)

Water that passes through the water-backs of the kitchen stove to the hot-water tank is particularly likely to collect iron, which accumulates at the bottom of the hot-water tank. This deposit may accumulate for days and even weeks until some unusual draught of water or other disturbance occurs—perhaps on washing day. When this happens it is very objectionable.

The household filter is the most convenient and satisfactory means of removing iron deposits from water that is otherwise good. The removal of iron from a city's water supply is a distinct process rarely combined with purification. In most cases iron may be removed by

thoroughly aerating the water in order to drive off the excess of CO_2 and in order to introduce oxygen necessary to oxidize the iron from the soluble ferrous state, in which it exists, to the insoluble ferric state. The precipitated ferric salts can then be removed by sedimentation or, better, by filtration.

LEAD

Tests.—The presence of lead may be discovered by chemical tests or surmised from the symptoms of lead poisoning among those who use the water. In the amounts present it does not affect either the appearance or taste of the water.

It is possible to determine the presence of lead in clear water and roughly estimate its amount by acidifying with acetic acid, saturating with hydrogen sulphid and comparing the brown tint produced with that produced by standard lead solutions contained in Nessler tubes similar to those for containing the sample under examination. This method is not applicable if the water is colored or contains iron—in this case special analytical procedures are necessary.

The sample of water used for testing lead should be the first portion (a pint or less) drawn after standing at least one hour in the pipes.

No water should be used for drinking purposes containing even a trace of lead, for, however minute it is, its presence in the water indicates danger. Very often the sample examined will not represent the daily maximum. For lead poisoning and its relation to water see p. 810.

EXPRESSION OF CHEMICAL RESULTS

Formerly results were expressed in grains per gallon. After the introduction of the metric system results were expressed in parts per 100,000, but now results are commonly expressed in parts per million. The latter method has the advantage that 1 milligram is .000,001 liter, and, therefore, 1 milligram in 1,000 c. c. = 1 part per million. A liter or a fraction thereof of the water to be analyzed is used, which greatly simplifies the calculations.

Of course, the assumption is made that a liter of water weighs a kilogram. This is sufficiently accurate for potable waters, but introduces an error where mineral waters are dealt with whose specific gravities are appreciably higher than unity. In such cases the water may be actually weighed, or else the weight may be estimated from the known specific gravity and volume.

The results expressed in parts per 100,000 or in grains per gallon

may be transformed to parts per million, or, conversely, by the use of the following table:

	Grains per U. S. gallon	Grains per Imperial gallon	Parts per 100,000	Parts per 1,000,000
1 grain per U. S. gallon	1.000	1.20	1.71	17.1
1 grain per Imperial gallon	0.835	1.00	1.43	14.3
1 part per 100,000	0.585	0.70	1.00	10.0
1 part per 1,000,000	0.058	0.07	0.10	1.0

CHAPTER III

MICROSCOPICAL EXAMINATION OF WATER

The chief object of the microscopic examination of water is the determination of the presence or absence of those microorganisms which produce objectionable tastes and odors. In certain cases the determination is also of value as an index of pollutions or as a guide to the identity of the water. The microscopical organisms comprise the Diatomaceæ, Chlorophyceæ, Cyanophyceæ, Fungi, Protozoa, Rotifera, Crustaceæ and other organisms minute in size, but not including the bacteria. Fragments of organic matter, broken-down organisms, zooglea, etc., should be termed amorphous matter. Clay, silt, oxid of iron, and mineral matter in general are not included under amorphous matter and are not measured by microscopic examination.

The term "microorganisms" as used by the water analyst includes all organisms, whether plant or animal, that are invisible or barely visible to the naked eye, other than bacteria. The bacteria are set apart, inasmuch as their significance and the method of studying them is different from all other microscopic organisms. As Whipple aptly phrases it, "Bacteria make a water unsafe, microorganisms make it unsavory."

Methods of Microscopical Examination.—The standard method of microscopical examination consists in concentrating a given quantity of water (250 or 500 c. c.) by filtration through sand. A straight sided cylindrical funnel two inches in diameter for the first nine inches, tapering to one-half inch in the next three inches, and concluding with a one-half inch tube two and one-half inches long, is used. A perforated rubber stopper (size No. 1) capped with a circle of silk bolting cloth is pressed tightly into the lower end. On top of this about five-eighths of an inch of prepared sand is poured, and the filter is then ready for use.

The sand used is quartz sand, washed and ignited, and of such a size that it will pass through a sieve having 60 meshes to the square inch, but will not pass a 120-mesh sieve.

When all the water has passed off and the sand begins to dry the stopper is removed, and the sand washed into a test tube with five cubic centimeters of distilled water from a pipette. The mixture of sand, organisms, and water is agitated and the supernatant suspension de-

canted into a second tube, from which one cubic centimeter is withdrawn and examined in a counting cell of that capacity, a specially ruled ocular micrometer and a ⅔-inch objective being used. This is the Sedgwick-Rafter process.

Significance of the Examination.—The microscopical examination of water is of great value in supplementing the chemical and bacterial analyses. It may explain the cause of odors and tastes in a water; it may explain certain chemical determinations, as albuminoid ammonia, dissolved oxygen, oxygen consumed, carbon dioxid, etc.; it may indicate sewage contamination; it may suggest the state of self-purification of a polluted water; it may identify the source of the water.

Several of the microscopic organisms, when present in sufficient quantities, give rise to objectionable odors and tastes, either when in a vegetative state or upon decomposition. The natural odors of organisms are due to oils analogous to the essential oils as in peppermint and in certain fishes. In general, the diatoms have an aromatic odor, increasing to that of a geranium leaf, and even to an intensity that is fishy. The cyanophyceæ, or blue-green algæ, have a grassy or moldy odor. The chlorophyceæ have little odor, although some of the motile forms give rise to faintly fishy odors. The ciliated protozoa have in general no odor. Uroglena, synura, dinobryon, and peridinium may and often do give rise to fishy odors. Of the other microorganisms, the rotifera and crustacea, no forms have been recorded as giving rise to objectionable odors. These forms are present only when there are lower forms upon which to feed. They are scavengers and as such may be considered as desirable elements in a water. Their presence, however, calls for an investigation of the nature of their food supply, as it is often furnished by pollution. This does not necessarily hold true in all cases.

Besides these animal and plant forms there may be present also sponges, mosses, yeasts, and molds, the significance of which is varied and dependent upon local conditions.

THE BACTERIOLOGICAL EXAMINATION

Practically all natural waters contain bacteria. This is true of rain water, ground water, and the waters of lakes, rivers, and oceans. The number and variety of the bacteria vary greatly in different places and under different conditions. The bacteria are washed into the water from the air, from the soil, and from almost every conceivable object. The intestinal contents of animals pollute waters with enormous numbers of microorganisms, but it is the infection with certain species from man that makes water most dangerous when consumed by his fellowmen.

THE NUMBER OF BACTERIA IN WATER

The number of bacteria is not as important as the kind, yet much may be learned from a simple enumeration of the bacteria. Roughly speaking, the number of bacteria in water corresponds to the amount of organic pollution. No known method furnishes a complete census of the bacterial population of a given sample of water. Methods based upon the direct microscopic count of the bacteria do not distinguish between the live and the dead ones; further, only those that may readily be seen by simple methods are thus visible. Many bacteria, especially those pathogenic for man, do not vegetate at 20° C., so that the usual counts upon gelatin may vary greatly from those obtained upon agar at 37° C. Some varieties require acid, others alkaline media; some are aerobic, others anaerobic; some will not grow unless the medium contains blood or other suitable pabulum, and so on through a wide gamut of conditions.

Although it is not possible to determine the total number, inferences of importance may be drawn from the differences in the numbers of bacteria in a given water obtained by different methods. Thus a water containing great numbers of bacteria, when counted upon gelatin at 20° C., and but few colonies upon agar at 37° C., has little sanitary significance, whereas a water containing few bacteria, but most of them varieties that grow upon agar at 37° C., with relatively few at 20° C., must be looked upon with suspicion. The distinction between polluted waters and waters of good quality is more sharply marked by counts at 37° C. than is the case with counts at 20° C. Another advantage of growing the plates at a higher temperature is that the results are available in a much shorter time.

The number of bacteria which grow at 40° C. are of special value, since this class includes the typhoid bacillus and other water-borne pathogens, but excludes the common water bacteria of little sanitary importance. The significance of acid-producing bacteria which grow at 40° C. upon litmus lactose agar is a well-known method in differentiating and determining the number of organisms belonging to the colon type in a water.

From Germany we have the arbitrary standard based upon the dictum of Koch that a good water should not contain over 100 bacteria per c. c. This is a good working rule, but should not be taken too literally. Thus, water may contain great numbers of the common aquatic bacteria which vegetate at room temperature and which are not harmful to man. Surface waters contain the greatest numbers on account of exposure to contamination to which they are liable; rain waters contain comparatively few, excepting the first shower through a very dusty at-

mosphere; ground waters from the depths are practically sterile. Unpolluted shallow well waters are also exceptionally free. The number and significance of the bacteria, therefore, vary with the source of the water. For example, a hundred bacteria, including a few colon bacilli, in a well water would be regarded with great suspicion, whereas a hundred or more bacteria, with an occasional colon bacillus, in a river water draining an uninhabited watershed would be normal.

The number of bacteria in water depends somewhat upon the manner in which it is stored. Thus a water containing a few organisms placed in a closed bottle and kept at room temperature may, at the end of 24 hours, contain hundreds or thousands per c. c. I once examined a deep well water that was practically sterile as it came out of the earth, but when stored in a cistern gave over a thousand organisms per c. c. These came from the multiplication of the bacteria that entered the water from the air, dust, leaves, and other sources. On the other hand, water stored in impounding reservoirs shows a marked diminution in the number of bacteria.

The numerical determination of bacteria in water is of very great value when studying surface waters, such as lakes and rivers. As a rule, the number of bacteria is proportional to the pollution of a river—not necessarily fecal pollution, but pollution from dead organic matter of one kind or another. The bacterial content of a river water varies sharply from time to time. Contrary to the usual opinion, a river contains more bacteria in the winter time than in the warm weather. During times of freshets or turbidity the number of bacteria will rise very abruptly. In other words, the number of bacteria in a stream is an index of its turbidity. It is an interesting fact that in the Potomac and other rivers the bacterial curve does not correspond to the typhoid fever curve. Typhoid in Washington is highest in summer, but the bacteria are most numerous in winter. While sudden variations in the number of bacteria have a ready explanation in the case of turbid and torrential rivers, in the case of lakes, and especially in a ground water, variation in numbers indicates nearby sources of pollution and is a danger signal. For shallow wells the interpretation of numbers is not so easy, largely because infection may enter at the surface. Wells which are poorly protected at the top will always show an unusually large number and variety of bacteria.

Numerical determination is also of importance in tracing imperfections and leaks in a water supply. Thus Dr. Shuttleworth, of Toronto, was able through this means to suspect a broken water main, and upon examination it was found that a whole section of the conduit had dropped out of place, so that the supply was being taken from the lake near the shore instead of some distance away where the intake was located.

The great value of the numerical estimate of bacteria is well known in determining the efficiency of filters.

Method for Determining the Number of Bacteria in Water.—Collection of the Sample.—For a complete physical, chemical, and microscopical analysis of water one gallon is necessary. If the sample has been collected in a sterile container with bacterial precautions, the same sample may serve for the bacteriological examination. Usually the bacteriological samples are collected separately in special bottles holding 2 ounces.

The bottles should be of hard, clear white glass and have a glass stopper. They should be chemically clean and sterilized at 160° C. for 1 hour, or in the autoclave at 115° C. for 15 minutes. For transportation they may be wrapped in sterile cloth or paper, but, better, the neck may be covered with tinfoil and the bottle placed in a tin box. When bacterial samples must of necessity stand 12 hours before plating, bottles holding more than 4 ounces should be used. Cork stoppers should never be permitted, except when physical or microscopical examination only is to be made. Earthen jugs and metal containers are entirely unsuited.

The water should be examined as soon after collection as practicable. Generally speaking, the shorter the time elapsing between the collection and analysis, the more reliable will be the analytical results. If too long a time intervenes, it affects especially the bacterial tests, for bacteria multiply enormously when water is kept in a bottle at ordinary temperature. The oxygen consumed, oxygen required, and nitrites are also materially affected by comparatively short delay.

Care should be taken to secure a sample which is thoroughly representative of the water to be analyzed. A pump should be operated five minutes, or water faucet allowed to run several minutes, before the bottle is filled. In collecting samples of surface waters the specimen should not be obtained too near the bank of the stream or pond. A note should be made as to whether the specimen is collected from the surface or at what depth under the surface it is taken. It is always advisable to take the temperature of the water at the time of collection.

Bacteriological Method.—The standard medium for determining the number of bacteria in water is a nutrient gelatin having a reaction of +1 per cent., using phenolphthalein as an indicator. The gelatin is made by using distilled water and an infusion of fresh lean meat, and not meat extract. The sodium chlorid is omitted. The medium contains 1 per cent. of Witte's peptone and 10 per cent. of gelatin.

The sample of water must be shaken vigorously at least 25 times in order to break up the bacterial clusters and to obtain a uniform suspension. If the water contains less than 200 bacteria per c. c., 1 c. c. of it may be placed directly in the petri dish, then add 10 c. c. of the

standard gelatin. Mix well, congeal, and incubate at 20° C. for 48 hours in a dark, well-ventilated incubator where the atmosphere is practically saturated with moisture. If there is reason to believe that the number of bacteria is more than 200 per c. c., dilute by mixing 1 c. c. of the sample with 9 c. c. of sterilized tap or distilled water. Again shake 25 times and plate 1 c. c. of the dilution. Higher dilutions may be made in 99 c. c. and so on. In the case of an unknown water or sewage it is customary to use several dilutions of the same sample. Count the colonies upon a Wolffluegel apparatus or a Jeffer's plate. A successful plate should contain not more than 200 colonies. The whole number of colonies on a plate should be counted, the practice of counting a fractional part being resorted to only in cases of necessity.

When agar is used for plating it will be found advantageous to use petri dishes with porous earthenware covers in order to avoid the spreading of colonies by the water of condensation.

In order to avoid fictitious accuracy and yet express the numerical results by a method consistent with the precision of the work, the table below should be followed in expressing the numbers of bacteria per c.c.:

From	1	to	50	recorded	as	found		
"	51	"	100	"	to	the	nearest	5
"	101	"	250	"	"	"	"	10
"	251	"	500	"	"	"	"	25
"	501	"	1,000	"	"	"	"	50
"	1,001	"	10,000	"	"	"	"	100
"	10,001	"	50,000	"	"	"	"	500
"	50,001	"	100,000	"	"	"	"	1,000
"	100,001	"	500,000	"	"	"	"	10,000
"	500,001	"	1,000,000	"	"	"	"	50,000
"	1,000,001	"	10,000,000	"	"	"	"	100,000

KINDS OF BACTERIA IN WATER

Water analysis is in its infancy so far as methods for determining the kinds of bacteria are concerned. It is comparatively easy to isolate colon bacilli and to determine their approximate number in a water. It is also comparatively easy to isolate cholera vibrio. Methods for determining whether a water does or does not contain typhoid, dysentery, and other pathogenic parasites are tedious, difficult, and often impossible in the present state of our knowledge.

A certain amount of information may be gleaned from the presence and number of organisms belonging to certain groups, such as chromogenic, liquefying, and fermenting types. Chromogenic organisms exist everywhere in surface waters. They should be practically absent from ground waters. The same is true of organisms that are able to liquefy gelatin and ferment sugars. The chromogenic, proteolytic and ferment-

ing types are widespread in nature and exist almost everywhere in the air, the soil, and in surface waters. Their presence in a ground water signifies contamination or pollution, often from the surface.

The significance of the various types of bacteria that grow at different temperatures has already been discussed.

THE COLON BACILLUS

The colon bacillus is very widely distributed in nature. Its normal habitat may be regarded as the intestines of man and many other animals. It even exists in some plants. The colon bacillus is usually taken as an index of pollution. The sanitary significance of colon bacilli in water varies with their number and, further, with their source. While the colon bacillus indicates pollution, it does not necessarily signify danger, that is, infection.

By common consent a ground water should be condemned if it contains even a few colon bacilli, for these organisms have no business in a soil-filtered and properly protected well or spring water. Surface waters are not regarded as particularly suspicious, provided they have not more than one colon bacillus per c. c., especially if the surface water is known to drain an uninhabited or controlled catchment area. Many of the colon bacilli in a surface water come from the droppings of wild and domestic animals and, therefore, are infinitely less indicative than those that come from the intestinal tract of man. The source of the colon bacillus can only be determined by an inspection of the watershed. A water containing 10 colon bacilli or more per c. c. should be regarded as grossly polluted and very likely to contain infection. Tests for the colon bacilli in water must, therefore, be qualitative and quantitative.

The absence of colon bacilli in water proves its harmlessness so far as bacteriology can prove it. It is fair to assume that typhoid bacilli, dysentery bacilli, and other intestinal parasites could not be present in a water in the absence of the colon bacillus. It is possible to conceive that in rare instances a water may be polluted with urine alone containing typhoid bacilli, but no colon bacilli.

Presumptive Tests for the Colon Bacillus.—Presumptive tests or partial tests are sometimes used to determine the presence of *B. coli.* These tests, while unreliable, sometimes afford useful information. They consist, as a rule, in planting small quantities of the water sample in lactose bile or lactose bouillon in fermentation tubes and incubating at 40° C. Under these circumstances it may be presumed that in the absence of fermentation colon bacilli are absent, and that fermentation with gas production indicates their presence. Both these conclusions may be misleading. Grossly polluted waters containing many colon bacilli may be slow in fermenting sugars with the production of gas on

account of the preponderance of other more active species. On the other hand, many organisms other than the colon bacillus often found in water ferment sugars with gas production. It is therefore necessary to isolate the suspected organism in pure culture and pass it through the well-known tests before it is labeled *B. coli.*

Qualitative Methods.—*Isolation.*—It is comparatively easy to isolate the colon bacillus in pure culture. Before an organism is labeled *B. coli* it should correspond to the following: It should be a relatively small, non-spore-bearing rod having rather sluggish or no motion; it should not liquefy gelatin; it should ferment dextrose broth with the formation of about 50 per cent. gas, one-third of which should be carbon dioxid and two-thirds hydrogen; it should coagulate milk, with the production of acid at 37° C., but without liquefaction of the coagulum. This coagulation should occur either spontaneously or on boiling; it should produce nitrite and indol in peptone solution and reduce nitrates.

To isolate *B. coli* the following method is satisfactory. Either plate the water directly upon lactose litmus agar and fish the red colonies, or plate directly upon Endo's medium and fish the red colonies. An enriching, and hence a surer, method, especially where there are very few colon bacilli in a water, is first to plant the water in fermentation tubes containing lactose or dextrose broth, incubate at 37° to 40° C. As soon as gas production is noted plate a small quantity of the growth upon lactose litmus agar or Endo's medium and study the red colonies for cultural and morphological characters.

The number of colon bacilli in a water may be determined by several methods:

(1) *The Fermentation Test.*—Add measured quantities of the water sample to fermentation tubes containing lactose broth. Ordinarily 0.1, 1.0, and 10 c. c. of water are used in this test. If the water is highly polluted smaller quantities, such as 0.01 or 0.001 of a cubic centimeter, should be used. If in such a series fermentation with gas production occurs in the tubes containing 1 c. c. or more, but does not take place in the tubes containing the smaller portions, it may then be stated that the water contains at least one colon bacillus per cubic centimeter, *provided* the isolation tests show that the fermentation was caused by this organism.

(2) The number of colon bacilli may be determined approximately by planting the water directly upon the surface of lactose litmus agar or Endo's medium. The red colonies should then be studied to determine how many of them are *B. coli,* and the number may thus be approximated per cubic centimeter.

SEWAGE STREPTOCOCCI

Fresh sewage from man and other mammalian animals usually contains streptococci resembling the *Streptococcus pyogenes*. These intestinal cocci, which are known as sewage streptococci, grow more readily than the pyogenic varieties, and produce a pinkish colony on lactose litmus agar at 37° C., by which their presence and number may be detected in water. These streptococci are not hardy, and therefore when found in a water represent immediate pollution. The general consensus of opinion is that the occurrence of these organisms in a water seems to be of less value than in the case of *B. coli*, and the streptococcus test is therefore of subordinate importance.

TYPHOID BACILLUS

The search for a typhoid bacillus in water is frequently like looking for a needle in a haystack. It is probable that the typhoid bacillus rarely, if ever, multiplies in natural waters. The dilution is usually enormous, and their number is therefore comparatively few. With modern methods and the use of Endo's medium it is comparatively easy to isolate typhoid bacilli from water richly seeded with these organisms, but it is practically a hopeless task to find them when there are only a few in a glassful. Great care must be exercised before an organism isolated from water is reported as *B. typhosus*. There are many organisms in water closely resembling typhoid, some of them even giving pronounced agglutination with specific serum. Thus *B. proteus, B. fluorescens*, and even *B. coli* sometimes agglutinate with typhoid serum and in higher dilutions than typhoid strains themselves. An interesting instance of this was found in our studies of the Potomac River water. Frost isolated an organism, the *"Pseudomonas protea,"* from the filtered Potomac River water which, during the months of August, September, and October, 1909, was more common than *B. coli*. This organism could not be found in the raw water, nor could it be found in a large number of stools examined, which points to a saprophytic existence. This organism may readily be distinguished from *B. typhosus*, in that it has different cultural characters, and further that animals injected with cultures of *Pseudomonas protea* develop agglutinins for this organism, but not for *B. typhosus*.

CHOLERA

The cholera vibrio may be detected in water by making a Dunham's solution of the water itself; that is, to a large quantity of the water sample add sufficient peptone to make a 1 per cent. solution, and render slightly alkaline with sodium carbonate. The water should be placed in

Erlenmeyer, Fernbach, or similar flasks, presenting a large surface favoring aerobic development. The flasks are then placed in the thermostat at 37° C. and in 16, 18, 24 hours, or longer, a loopful of the surface growth is planted upon agar, Endo's medium, or gelatin. Cholera colonies upon gelatin have a ground-glass appearance when examined under a low power of the microscope, with irregular margins, and the gelatin is slowly liquefied. Upon agar the colonies are not particularly distinctive; upon the surface of Endo's medium cholera grows as faintly pinkish, moist, translucent colonies, not unlike typhoid colonies, excepting that they have slightly more color. Dependence cannot be placed upon the appearance of the colonies nor upon the morphological characteristics of the organism. Suspicious colonies should be isolated and tested with an agglutinating serum of known specificity having a high agglutinating value. All organisms that are agglutinated with this serum in a dilution of 1-1,000 or over may be regarded as cholera. This, however, should not be accepted as final, for, as is the case with typhoid, there are numerous cholera-like organisms in water that agglutinate with a cholera serum, but which upon further study have characteristics which plainly show that they are not the organism which causes cholera. Final dependence should be placed upon Pfeiffer's phenomenon and upon cross-agglutinating tests or absorption tests to eliminate the phenomenon of group agglutination.

CHAPTER IV

INTERPRETATION OF SANITARY WATER ANALYSIS

The interpretation of a sanitary water analysis is much more difficult than the analysis itself, where everything may be carried out by rule of thumb in accordance with standard procedures. Single or occasional determinations of either the chemical or bacterial properties of water are of little value. A single water analysis is often misleading, especially in surface waters, which may vary greatly from time to time. A river water may require repeated examinations extending over long periods of time correlated with conditions of rainfall, stream flow, wind, temperature, sewage pollution, and other factors in order to be helpful.

There have been much conflict and useless discussion between chemists and bacteriologists concerning the relative advantages of their methods. The chemists were first in the field, but the limitations of chemical methods were strongly emphasized when it was shown that chemistry can only indicate pollution but cannot discover infection. Much was hoped from bacteriology, but it is rather exceptional that bacteriologists are able to isolate pathogenic microorganisms from a sample of water. For the most part, the routine bacteriological examination of water does nothing more than the chemical examination, that is, it shows pollution but does not prove infection. Both chemical and bacterial analyses of water have, therefore, distinct limitations; they do not antagonize, but supplement each other. From the chemical side we learn much of the past history of a water; the bacteriology tells us more of its present state. Chemical methods reign supreme when we desire to discover the presence of lead or other inorganic poisons; also in determining the hardness, mineral and organic constituents, etc. From the number and character of the bacteria in water we obtain a fair index of the presence and degree of pollution. Occasionally bacteriologists may determine whether a water contains certain specific agents, such as cholera vibrio. It must, however, be admitted that the ordinary routine chemical and bacterial examination of water affords but meager information, especially when only one analysis has been made. Fortunately, the inferences drawn from a sanitary water analysis

are on the safe side, as many good waters are condemned, so that it would be very difficult for an unsafe water to pass the muster of a complete sanitary analysis. At most, the information furnished is only of present conditions and is not a guarantee of future safety. A surface water or a ground water may to-day be exceptionally free from chemical impurities and practically sterile bacteriologically, whereas to-morrow it may contain typhoid, dysentery, cholera, or other water-borne infections; these may come from sources that would at once be perfectly evident from a sanitary survey of the watershed.

A sanitary survey of the catchment area is frequently of much greater practical importance than all the information furnished by the laboratory. It needs neither chemists nor bacteriologists to tell us that the water from a creek with an overhanging privy a short distance above will some day carry infection; or that the water from a shallow well in limestone or coarse gravel very near a leaking cesspool must be a source of danger. A sanitary survey is able to discover the sources of contamination, the kinds of pollution, and the degree, often with greater precision than combined chemical or bacteriological tests. No sanitary analysis of a water can therefore be considered complete unless it includes an examination of the watershed and a study of the geology and topography of the catchment area.

From a sanitary standpoint, the principal substances to look for in a chemical analysis are the organic matter, nitrates, nitrites, and chlorin. Of these the nitrites are the greater danger signal, indicating oxidation of organic matter through bacterial activity. High chlorin and nitrates without nitrites indicate passed or remote pollution; this is a frequent combination in ground waters. The ammonias (free and albuminoid) are a measure of the amount of nitrogenous organic matter in the water. A surface water may safely contain an amount of albuminoid ammonia that would be suspicious in a ground water. The significance of the chlorin varies with the location and source of the water. Ground waters should contain fewer bacteria than surface waters. Artesian wells should be practically sterile, and a good surface water should not contain over 100 bacteria per cubic centimeter. Waters that vary in composition from time to time without evident cause must be regarded as unsafe. This applies particularly to ground waters. Surface waters vary greatly as the result of freshets, etc., but a ground water, pond, or lake should show no such sudden variations.

These general statements may be quite misleading when interpreting the analysis of a specific case. Thesefore several selected analyses and interpretations have been given below.

Allowable Limits.—The following are sometimes considered as the allowable limits of the impurities commonly regarded as permissible in drinking water:

Free ammonia ..... 0.015-0.03 part per million
Albuminoid ammonia ..... 0.07 -0.35 " " "
Nitrogen as nitrites.....None, or at most a trace (0.0004)
Nitrogen as nitrates..... 0.16 -0.3+

Chlorin depends upon the normal chlorin content of unpolluted surface waters in the neighborhood.

Bacteria not over 100 per cubic centimeter.

Colon bacillus should be absent from the ground water. Not more than 1 per 10 c. c. in a stream or in a river water.

These figures must not be taken literally, and are not given as standards of purity, but the maximum limits of the impurities allowable under ordinary conditions. It will be seen from the illustrative analyses given below that at times these limits may be exceeded without sanitary significance, whereas at other times a water well within the prescribed limits may contain infection.

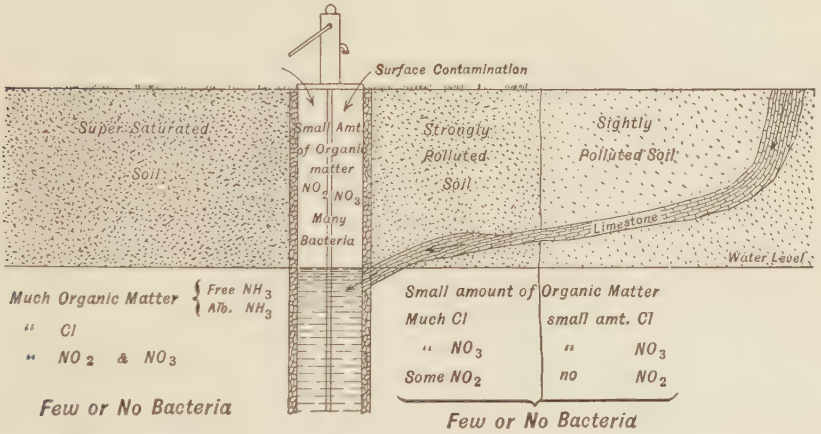


FIG. 104.—DIAGRAM ILLUSTRATING THE CHARACTER OF THE GROUND WATER IN RELATION TO SOIL POLLUTION, TO ASSIST THE INTERPRETATION OF A SANITARY ANALYSIS. (See also Nitrogen Cycle, page 676.)

For a better understanding a number of sanitary analyses of water are given with an interpretation. The student is advised first to study the analyses, draw his own conclusions, and then compare them with the interpretation given.

ANALYSIS NO. 1—*Gross Pollution*

Free ammonia	0.214	part per million
Albuminoid ammonia	0.810	" " "
Nitrogen as nitrites.....	0.005	" " "
Nitrogen as nitrates.....	21.0	parts " "
Chlorin	47.0	" " "

Total residue	412.0	parts	per	million
Volatile residue	279.0	"	"	"
Fixed residue	133.0	"	"	"

Bacteria per c. c. upon gelatin at 20° C..... 65,000
Bacteria per c. c. upon agar at 37° C..... 120,000

Many liquefying colonies. Many chromogens per c. c. Fermentation in lactose bouillon in 0.001 c. c. *B. coli* present in 0.01 c. c.

This represents a grossly polluted water and should unhesitatingly be condemned, no matter what its source.

The following analysis of the Hamburg public supply from the Elbe River during the cholera epidemic of 1892 is given in *Chemical News,* LXVI, 144:

Analysis No. 2—*Elbe River During Cholera Epidemic*

Appearance	Turbid and very yellow.		
Taste	Slightly unpleasant.		
Odor	Extremely little.		
Deposit	Small and dirty-looking.		
Chlorin	472.0	per	million
Free ammonia	1.065	"	"
Albuminoid ammonia	0.293	"	"
Nitrates	26.43	"	"
Required oxygen (15 minutes)	0.928	"	"
Required oxygen (4 hours)	3.428	"	"
Total solids	1,160.7	"	"

This is given simply as an instance of a grossly polluted river (Elbe) water, known to be infected.

Analysis No. 3—*Boston Tap, Typical* (not averaged results)

Free ammonia	0.010	part	per	million
Albuminoid ammonia	0.114	"	"	"
Nitrogen as nitrites	0.000	"	"	"
Nitrogen as nitrates	0.02	"	"	"
Chlorin	2.7	parts	"	"
Total residue	27.0	"	"	"
Volatile residue	10.0	"	"	"
Fixed residue	17.0	"	"	"

Hardness, 13°.

Bacteria per c. c. upon gelatin at 20° C..... 77
B. coli ..... None

This is a surface water, collected in impounding reservoirs and stored about 30 days before it reaches the consumer. The watershed

is fairly well protected. The chemical analysis shows little organic pollution; the ammonias are moderate in amount, nitrites absent; nitrates low; chlorin normal; bacteria indicate nothing suspicious. The water is of good sanitary quality, judged by chemical and bacterial analysis.

ANALYSIS NO. 4—*A Suspicious Water*

Free ammonia	0.018	part	per	million
Albuminoid ammonia	0.020	"	"	"
Nitrogen as nitrites	0.007	"	"	"
Nitrogen as nitrates	1.5	parts	"	"
Chlorin	19.3	"	"	"
Total residue	106.0	"	"	"
Volatile residue	37.0	"	"	"
Fixed residue	69.0	"	"	"

Hardness, 33.8°.

(The residue did not char and gave no odor upon ignition.)

Bacteria per c. c. upon gelatin at 20° C.	60
Bacteria per c. c. upon agar at 37° C.	45

No liquefying colonies. One chromogen per c. c. No fermentation in lactose bouillon in 10 c. c. No *B. coli.*

This water came from a driven well at Wenham, Mass. Upon inspection it was found that the well was 400 feet from a stable, 200 feet from a cesspool, and 250 feet from the house.

The first thing that strikes our attention in this analysis is the high chlorin. This, however, lacks sanitary significance, as it is normal for the ground waters of this neighborhood. The hardness of the water is due to the very fertile character of the surrounding soil through which the water percolates. The carbonic acid taken up by the water from the decomposing organic matter dissolves the lime in the soil. The organic matter as represented by the ammonias is quite low. The nitrates are high and indicate that the water has dissolved this end product of the oxidation of organic matter in its passage through the soil and perhaps in seepage from the cesspool. The noticeable quantity of nitrites indicates that all the organic matter has not been consumed and that the mineralization is not complete. The small number of bacteria present shows that the filtering action of the soil through which the water passes is effective in keeping out sewage contamination either from the surface or from the cesspool. This conclusion is strengthened by the absence of fermenting organisms and especially the absence of *B. coli.* The absence of liquefying bacteria and the presence of an occasional chromogenic organism indicate that there is little or no contamination from the surface and, in fact, upon inspection the platform covering the well was found to be tight and well constructed.

This particular sample of well water shows nothing injurious to health, and if subsequent analyses are equally satisfactory the water may be used without fear for drinking purposes. It is plain, however, that this well needs watching, for it is evident that the soil is already surcharged with organic matter, some of which appears in the water, and a further loading of the soil or a break in the cesspool might readily infect the well.

ANALYSIS NO. 5—*Surface Pollution of a Well*

Free ammonia	0.022	part	per	million
Albuminoid ammonia	0.035	"	"	"
Nitrogen as nitrites	0.007	"	"	"
Nitrogen as nitrates	1.0	"	"	"
Chlorin	19.0	parts	"	"
Total residue	356.0	"	"	"
Volatile residue	151.0	"	"	"
Fixed residue	205.0	"	"	"

(Residue charred upon ignition with disagreeable odor.)

Bacteria per c. c. upon gelatin at 20° C.	9
Bacteria per c. c. upon agar at 37° C.	275

Many liquefying colonies. Several chromogens per c. c. Fermentation in lactose bouillon in 0.1 c. c. *B. coli* present in 1 c. c.

This is a shallow well in Washington, D. C., 28 feet deep, the water standing 4 feet in the well. There is a sewer 60 feet from the well and a privy within a block. The pump is old and of wood and the cover to the well is rotten at the base.

Although this water contains a small amount of organic matter, as indicated by the ammonias, every other factor indicates pollution both present and past. The nitrates and nitrites are high; the chlorin is excessive. It is important to notice that this water has only 9 bacteria per cubic centimeter when judged by the colonies that grow upon gelatin at 20° C. Nevertheless, it contains colon bacilli in 1 c. c., other fermenting organisms, as well as liquefying and chromogenic colonies. It is probable that most of the contamination in this case came from the surface, as the well had a very poor and leaky platform. This water should not be used for domestic purposes, and if it did not materially improve after the correction of the platform it should be condemned.

ANALYSIS NO. 6—*Well Water, Surface Pollution*

Free ammonia	0.007	part	per	million
Albuminoid ammonia	0.018	"	"	"
Nitrogen as nitrites	0.0005	"	"	"

Nitrogen as nitrates	2.5	parts per million
Chlorin	14.0	" " "
Total residue	62.0	" " "
Volatile residue	32.0	" " "
Fixed residue	30.0	" " "

(Residue charred upon ignition and gave disagreeable odor.)

Bacteria per c. c. upon gelatin at 20° C	820
Bacteria per c. c. upon agar at 37° C	640

Many liquefying colonies. Many chromogens per c. c. Fermentation in lactose bouillon in 1 c. c. *B. coli* in 10 c. c.

This water came from a shallow well in Washington, D. C., 18 feet deep, the water standing 3 feet from the bottom. The rather high nitrates and chlorin in this case represent past pollution. The small amount of organic matter with a trace of nitrites plus the number and character of the bacteria indicates surface pollution. This view is strengthened by the fact that repeated examinations of the water from this well showed marked variations in the number of bacteria. Upon inspection the pump and covering to the well were found in very bad condition, leaky, and with surface drainage toward the well.

Analysis No. 7—*Illustrating Remote Pollution*

Free ammonia	0.006	part per million
Albuminoid ammonia	0.011	" " "
Nitrogen as nitrites	trace	" " "
Nitrogen as nitrates	20.0	parts " "
Chlorin	89.0	" " "
Total residue	430.0	" " "
Volatile residue	113.0	" " "
Fixed residue	317.0	" " "

(No charring upon ignition; odor of burning rubber.)

Bacteria per c. c. upon gelatin at 20° C	92
Bacteria per c. c. upon agar at 37° C	16

No liquefying colonies. No chromogens per c. c. No fermentation in lactose bouillon in 10 c. c. *B. coli* absent.

This is a ground water from a shallow well in Washington, D. C. The well is 29 feet deep and the water stands 4 feet from the bottom. Top is well protected, waste water drains to sewer nearby. There are two privy vaults within two blocks of the well.

The analysis shows high chlorin and nitrates; otherwise nothing suspicious. This means remote pollution. The organic matter has been completely mineralized and the bacteria held back by the soil.

ANALYSIS No. 8—*High Chlorin*

Free ammonia	0.016	part	per	million
Albuminoid ammonia	0.015	"	"	"
Nitrogen as nitrites	0.000	"	"	"
Nitrogen as nitrates	0.14	"	"	"
Chlorin	11.20	parts	"	"
Total residue		"	"	"
Volatile residue		"	"	"
Fixed residue		"	"	"

Bacteria per c. c. upon gelatin at 20° C. 48
Bacteria per c. c. upon agar at 37° C. 12

No liquefying colonies. No chromogens per c. c. No fermentation in lactose bouillon in 10 c. c. *B. coli* absent.

This water is from a driven well at Beverley, Mass. The analysis shows nothing suspicious, excepting the high chlorin, which is normal for this neighborhood and therefore lacks sanitary significance.

ANALYSIS No. 9—*High Free Ammonia; Deep Well*

Free ammonia	0.170	part	per	million
Albuminoid ammonia	0.000	"	"	"
Nitrogen as nitrites	trace	"	"	"
Nitrogen as nitrates	0.0	"	"	"
Chlorin	3.1	parts	"	"
Total residue	115.0	"	"	"
Volatile residue	45.0	"	"	"
Fixed residue	70.0	"	"	"

No bacteria per c. c. upon gelatin at 20° C.
No bacteria per c. c. upon agar at 37° C.
No fermentation in lactose bouillon.

This water is from a driven well in Washington, D. C.; 96 feet deep, water stands 81 feet from the bottom. Good platform and drain, and pump is in first-class condition.

It is exceptionally pure, both chemically and bacteriologically, excepting the large amount of free ammonia. This supposedly comes from the reduction of nitrates.

It is not uncommon to find water from deep wells to be high in free ammonia, and it is assumed that this comes from a chemical reduction under high pressure, and perhaps temperature of the nitrogenous matter in coal and alluvial deposits.

ANALYSIS No. 10—*Rain Water Stored and Polluted*

Free ammonia	1.050	parts	per	million
Albuminoid ammonia	0.175	"	"	"

Chlorin	2.0	parts per million.
Nitrogen as nitrites	strong trace	" " "
Nitrogen as nitrates	0.0	" " "
Required oxygen	2.25	" " "
Total residue	20.0	" " "

Bacteria per c. c. upon gelatin at 20° 625

No fermenting organisms. No *B. coli.*

This is rain water from a dirty cistern. In appearance the water was clear and good. The analysis shows that the water is dirty and contaminated with organic matter. The bacteriological results indicate absence of fecal pollution. The water is undesirable, but not dangerous, as far as infection is concerned.

ANALYSIS No. 11—*Artesian Well Water, Showing the Effects of Storage*

(The figures are in parts per million)

	Water Directly from the Well	Same Water from the Storage Cistern
Free ammonia	.052	.062
Albuminoid ammonia	.003	.016
Nitrogen as nitrites	.000	.0007
Nitrogen as nitrates	.01	.01
Chlorin	10.4	10.2
Dissolved oxygen	10.65	10.69
Oxygen required	.10	.15
Total residue	111.0	97.
Volatile residue	40.0	30.
Fixed residue (mineral matter)	71.0	67.
Bacteria per c. c. upon gelatin at 20° C.	6	6500
Fermentation in lactose bouillon in	none	in 0.1 c. c.
B. coli	absent	absent

This water is from eight artesian wells at the Government Hospital for the Insane at Anacostia, D. C., 375 feet deep. The water is forced out by compressed air and flows by gravity to the storage cistern, which is of brick and cement, and has a capacity of 80,000 gallons.

It will be observed that this water is low in total solids and is almost free of organic matter as represented by the ammonias, nitrites, nitrates, and oxygen required. The water is clear as it flows from the ground, but soon turns slightly yellowish on account of a small amount of iron in the ferrous state that is oxidized to the ferric salt, which is insoluble and is precipitated upon contact with the air. The amount of chlorin is somewhat large, but has no sanitary significance in this case. The principal point in this analysis is the bacteriology, which shows the water to be practically sterile as it flows from the ground, but which contains over 6,000 bacteria per cubic centimeter in

ANALYSIS No. 12—*Chemical and Bacteriological Changes in Potomac River Water as the Result of Storage and Filtration*

(The figures are the averages of fourteen representative analyses)

	Dalecarlia Inlet Raw Water Entering Storage Reservoir	*Dalecarlia Outlet* Raw Water After About 3 Days' Storage	*Georgetown Reservoir* Second Storage Reservoir (Water remains here about a day)	*Washington Reservoir* 3rd Storage Basin Water Applied to Filter	Filtered Water from Filtered Water Reservoir
Free ammonia	0.024	0.027	0.022	0.017	0.015
Albuminoid ammonia	0.161	0.131	0.117	0.096	0.054
Nitrogen as nitrites	0.0031	0.0051	0.0065	0.0056	0.0003
Nitrogen as nitrates	0.61	0.57	0.6	0.61	0.67
Chlorin	2.6	2.61	2.61	2.47	2.53
Total residue	203.0	163.0	160.0	141.0	127.0
Volatile residue	47.1	48.0	49.0	41.0	39.0
Fixed residue	156.0	115.0	111.0	100.0	88.0
Bacteria per c.c. upon gelatin at 20° C	526	381	306	235	36
Per cent. of *B. coli* in 1 c.c.	42	40	33	16	4.7
Per cent. of *B. coli* in 10 c.c.	28	40	40	41	9.5
Total per cent. showing *B. coli*	71	80	73	52	14.2

the storage cistern. These come from the air and other contaminating objects, and illustrate the great growth of the common water bacteria in water stored under these circumstances. The slight increase of the ammonias and nitrites in the cistern water, as compared with the water direct from the well, indicates organic pollution and bacterial activity. The diminution in the residue results largely from separation of the iron. This water is pure and wholesome, despite the fact it contains many more bacteria than that usually allowed. It has been used for some years by about 3,000 persons, who are singularly free from typhoid fever and other water-borne diseases.

Analysis No. 12 is a good illustration of the bacteriological and chemical character of a river water, and illustrates the changes that occur during short storage (3 to 5 days) and after filtration through a slow sand filter.

It will be seen from this table that there is a gradual diminution in the amount of free ammonia and a more marked diminution in the amount of albuminoid ammonia. The amount of organic matter as represented by the ammonias is diminished just one-third. The nitrites show an increase during storage of the water, indicating active oxidation, but a marked decrease after it is filtered, showing the rapid completion of the oxidation of the organic matter in the filter. The nitrates show a tendency to increase in amount, which would be expected as the nitrites diminish. It is evident that storage and filtration have little effect upon the chlorin content of the water. The total residue diminishes as the result of storage, sedimentation, and filtration. It will be noted, however, that this diminution is more marked with the fixed residue than with the volatile residue.

The number of bacteria decrease as the result of storage, but the most marked decrease occurs as the result of filtration. It should be remembered that all the bacteria in the filtered water do not represent those which have passed the filter. The effect of the few days' storage upon this water does not very materially affect the number of *B. coli*, but there is a marked diminution in their number as the result of filtration.

Analysis No. 13—*Typical Analysis of Surface Waters*

The analyses of surface waters, shown in the table on page 774, with diagram showing the locations from which samples were obtained, will repay careful study. This table and diagram were furnished through the kindness of Professor Whipple.

TYPICAL ANALYSIS NO. 13 OF SURFACE WATERS

Harvard University Laboratory of Sanitary Engineering.

	Sample Number	Time Collection: Date	Time Collection: Hour	Description of Sample	Name of Collector	Physical: Temperature	Physical: Turbidity	Physical: Color	Physical: Odor
a	A	July 1	1 P. M.	Upland stream	John Doe	68° F.	2	12	1v
b	B	July 1	2 P. M.	Stream below cultivated land	John Doe	68° F.	4	18	2v
c	C	July 1	3 P. M.	Stream from swamp	John Doe	66° F.	3	125	3v
d	D	July 1	4 P. M.	Lower end of reservoir (surface)	John Doe	72° F.	10	55	4g
e	E	July 1	4 P. M.	Lower end of reservoir (bottom, 60 ft.)	John Doe	50° F.	15	250	4m
f	F	July 1	5 P. M.	Stream above city	John Doe	68° F.	2	5	2v
g	G	July 1	6 P. M.	Stream below city sewer	John Doe	71° F.	8	10	3d
h	H	July 1	5:30 P. M.	Sewage (fresh condition)	John Doe	75° F.	220	18	4d

Chemical Analysis (Parts per Million)

	Residue: Total	Residue: Loss on Ignition	Residue: Fixed	Suspended Matter: Total	Suspended Matter: Loss on Ignition	Suspended Matter: Fixed	Nitrogen: Kjeldahl	Nitrogen: Albuminoid Ammonia	Nitrogen: Free Ammonia	Nitrogen: Nitrites	Nitrogen: Nitrates	Oxygen Consumed	Chlorin	Total Hardness	Alkalinity	Incrustants	Magnesium	Iron	Fats	Free CO_2	Dissolved Oxygen: Parts per Million	Dissolved Oxygen: Per Cent. of Saturation
a	29	10	19	..	..	..	...	.056	.006	.001	0.25	2.4	2.3	12.0	10.0	2.0	....	0.1	..	2	9.19	100
b	42	16	26	..	..	..	...	.080	.020	.002	0.70	3.2	2.5	16.0	11.0	5.0	....	0.2	..	2	9.19	100
c	51	30	21	..	..	..	...	.250	.032	.001	0.50	19.6	2.4	10.0	9.0	1.0	....	0.4	..	8	8.92	95
d	47	22	25	..	..	..	...	.384	.058	.003	0.10	12.0	2.6	13.0	10.0	3.0	....	0.3	..	—2	8.81	100
e	83	45	38	..	..	..	...	.160	.182	.001	0 05	18.0	2.6	14.0	11.0	3 0	....	3.2	..	25	0	0
f	42	8	34	..	..	..	...	.046	.008	.000	0 20	3 0	2.2	27.0	20 0	7.0	4.0	0.1	..	2	9 19	100
g	92	22	70	..	..	..	...	.180	.094	.007	1.90	10.0	5.7	50.0	38.0	12.0	9.0	0.5	..	8	6.22	70
h	610	260	350	208	89	119	9.1	....	7.500	.210	0.30	53.0	68.0	85.0	60.0	25.0	15 0	1.5	25	25	0.85	10

	Bacteriological: Number per c.c.	Bacteriological: Test for *B. coli*: .001 c.c.	.01 c.c.	0.1 c.c.	1.0 c.c.	10 c.c.	Microscopical: Total Organisms per c.c.	Microscopical: Principal Genera per C.C.: Anabæna	Anthophysa	Crenothrix	Ciliata	Leptothrix	
a	40			0	0	0	10						
b	210			0	0	+	20						
c	420			0	0	+	105	60	30				
d	90			0	0	+	2,480	2,100	5				
e	800			0	0	0	300			200			
f	50			0	0	0	12						
g	3500			+	+	+	360				40	190	
h	2—	+	+	+	+	+	70				30		

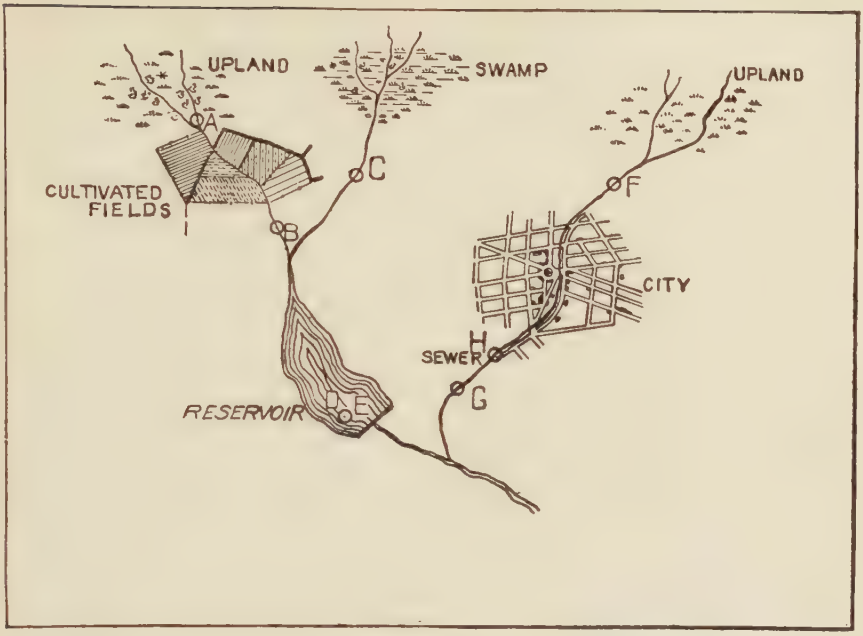


Fig. 105.

The diagram above (Fig. 105) shows the location of the samples employed in Analysis No. 13.

CHAPTER V

THE PURIFICATION OF WATER

The ways in which water may be purified for practical purposes are not many. It is worth noting that most of the advances in water purification come from the development of old empiric processes. It is only at long intervals that a new method or principle of treatment is discovered that is important enough to find a permanent place in the art of water purification.

The principal methods at present serviceable for the purification of water upon a large scale are: (1) storage, (2) filtration, (3) chemicals, such as ozone, hypochlorite of lime, sulphate of aluminium or iron.

No method of purifying water can be considered to approach a satisfactory hygienic standard that does not first of all practically eliminate water-borne diseases. The process must also reduce the turbidity and color to inappreciable amounts and remove something like 99 per cent. of the bacteria, when these organisms result from sewage pollution and are fairly numerous: there is perhaps no final reason for the bacterial standard. It has been adopted by consent because it represents a purification that is reasonably satisfactory and that can be accomplished at the small cost of about $10.00 per million gallons of water treated. With the further awakening of the sanitary conscience of the community the standards will inevitably tend higher, and it is probable that in time our standards will approach an ideal that is now not regarded as necessary. At present there is no evidence that the few microorganisms left in the water after a satisfactory method of purification, such as slow sand filtration, are injurious. Certainly, if injurious influence is exercised, it is too small to be determined or measured by any methods now at our disposal.

NATURE'S METHOD OF PURIFYING WATER

In nature, water is purified by various methods, the chief of which are: (a) evaporation and condensation, which makes rain water the purest of natural waters; (b) the self-purification of running streams, which is a variable and uncertain quantity; (c) storage in lakes and

ponds which clarifies water and in time eliminates danger; and (d) the physical, chemical, and biologic action of the soil upon water that filters into the earth, which is one of nature's greatest purifying agencies.

Evaporation and Condensation.—The purifying action of the distilling and condensing process through which all meteoric water passes is one of nature's beneficent processes. Enormous quantities of sea water, marsh water, and polluted waters of all kinds are thus returned to us suitable for domestic use. Somerville estimates that "186,240 cubic miles of water are annually raised from the surface of the globe in the form of vapor, chiefly in the intertropical seas." Water is thus constantly being purified in nature. The ocean has been compared to a boiler, the sun to a furnace, and the atmosphere to a vast still. The cooler air of the higher atmosphere and of colder zones acts as a condenser, causing the precipitation of the distilled water as rain. About three-fourths of the earth's surface (145,000,000 square miles) is covered with water, much of which is in the tropical belt.

Self-purification of Streams.—The self-purification of streams needs special discussion. Streams become purer during the course of their flow. Of this there can be no doubt. This half-truth based upon chemical data has in the past suffered sanitarians to permit the use of water that now we know was responsible for much sickness and many deaths. Streams become purer, but not pure. Some impurities always remain, that is, the process is not complete and final. All surface supplies are now regarded with suspicion and are either stored, filtered, or otherwise purified before they are used by educated communities.

It was formerly said that a stream purifies itself in seven miles. Such a generalization is absurd. We now know that it is not the distance so much as the time and opportunity for the various factors involved to become effective. Thus, Buffalo's sewage flows to Niagara's intake, a distance of about 16 miles, in a few hours. There is little chance for self-purification to take place, and despite the great dilution the danger is very great. Niagara's average typhoid rate for 10 years, from 1899 to 1908, was 132.9 per 100,000, the highest in the country. On the other hand, we have the following facts:

A good instance of the self-purification of streams resulted from the studies of the Potomac River and its relation to typhoid fever in the District of Columbia. The Potomac River drains an area of about 11,400 square miles which, in 1900, contained a population estimated to be about half a million, or about 44 per square mile. The velocity of the flow of the Potomac is extremely variable. It takes from 4 to 7 days for the water to travel from Cumberland to Great Falls (where the Washington intake is located), a distance of about 176 miles. The waters of the Potomac are directly polluted by sewage at numerous

points. The direct pollution is contributed by about 45,000 individuals, or 91 per cent. of the total population on the watershed. Of this pollution about 80 per cent. enters the river at points 176 or more miles from the intake at Great Falls, about 15 per cent. enters it at points between 50 and 170 miles above Great Falls, and 5 per cent. is contributed by about 2,200 of the population and enters the river at points between 19 and 50 miles above the intake. There is practically no direct pollution of the Potomac within 19 miles of the intake. Here we have an instance of a stream draining an extensive and populous area and receiving industrial and human wastes from many thousand persons. Nevertheless, self-purification has occurred to such an extent that little, if any, of the typhoid fever occurring in Washington could be attributed to the use of this water.

The Mississippi River is perhaps one of the best examples of the self-purification of a stream, for, after draining the entire continent of the United States in a flow of over 15,000 miles, it is exceptionally free of pathogenic bacteria at New Orleans, judged by the absence of colon bacilli.

The principal factors concerned in the self-purification of water are varied and interesting. They are: (1) Chemical, the oxidation of nitrogenous organic matter, resulting in its reduction or mineralization. (2) Biologic, the death of microorganisms through symbiosis, time, and various means; and (3) physical, such as dilution, sedimentation, sunlight, etc.

Oxidation.—Organic matter is gradually oxidized, thus diminishing the amount of food for bacteria. The activity of the oxidation depends largely upon the amount of dissolved oxygen in the water. It is therefore favored by falls, rapids, and a turbulent flow. It is mainly the aerobic bacteria which have an active proteolytic action, and are thus able to digest and destroy organic matter. During the course of flow the complex nitrogenous substances are thus mineralized. Chemical analyses show a rapid decrease in the amount of organic matter and an increase of nitrates, and diminution of nitrites. It was these facts that led chemists to conclude that flowing rivers soon purified themselves.

Biological Factors.—Minute animals such as infusoria, amebæ, water-worms, water-fleas, etc., which exist in countless numbers in certain waters, feed upon the organic matter and bacteria, and are a considerable factor in the self-purification of water.

Time and *symbiosis* play an important rôle with self-purification of streams, as they do elsewhere. Pathogenic bacteria die more quickly in a polluted water than in a pure water. It is probable that symbiosis here plays a part. The saprophytic bacteria somehow help to kill off the dangerous varieties. Pettenkofer believed that the greater part of self-

purification is due to the growth of algæ and other low forms of vegetation which clear the water of its impurities in the same way that the higher plants utilize the decomposing manure on cultivated fields. This view is endorsed by Bokorny, Emerisch, and Brüner and others who have studied the question. It is proven that these plants take up all manner of organic substances. This includes volatile fatty acids, amino acids, glucose, and urea. The purifying effects of water vegetation are therefore placed near the head of the list of self-purification.

Dilution and Sedimentation.—Dilution is one of nature's real sanitary blessings. The superabundance of water and air quickly dilutes the impurities under ordinary conditions so as to render them harmless. A small amount of infection in a great volume of river or lake water soon becomes so diluted as literally to become lost. It is true that one germ may cause disease just as a spark may start a forest fire, but the conditions must be exceptionally favorable. It is fortunate for us that a single typhoid, cholera, or dysentery bacillus, especially when attenuated, will not, as a rule, induce disease. It is further clear that the chances of receiving a single bacillus in the few glasses of water one drinks are mathematically very small when the dilution is very great. Owing to these facts and to the further fact that pathogenic spore-free bacteria soon become attenuated and die in water, dilution becomes one of our chief sanitary safeguards.

Sedimentation is favored by a slow-moving stream containing insoluble inorganic particles such as clay. In muddy streams such as the Mississippi and Potomac Rivers the water is purified in very much the same way that the snow clears the air. The particles, constantly settling, wash the water by enmeshing the bacteria, which are thus carried to the bottom, where they are imprisoned and die. It is almost a filtration process. The water is swept or scoured many times by the innumerable fine particles in a turbid stream. This is the same principle used to clarify water with chemical coagulants such as sulphate of alumina.

Storage in Lakes and Ponds.—Nature makes use of the purifying power of time in storing water in lakes and ponds and other surface collections. Very few parasites pathogenic for man multiply in water under natural conditions. In time they all die out. Hence a stored water is reasonably safe. In addition, the organic matter undergoes decay and returns to its simple mineral constituents. Hence a stored water will in time free itself not only of harmful parasites, but also of most of its organic pollution. The stagnation of stored water has been described on page 706.

The purifying power of the soil has been fully discussed in connection with the nitrogen cycle (page 676).

Sunlight.—The germicidal influence of sunlight exerts its power

upon all surface waters. The depth of penetration, however, varies with the turbidity of the water, the strength and direction of the sun's rays, and other factors.

DISTILLED WATER

The distillation of water is the only method known for rendering it pure in a chemical sense. From a hygienic standpoint it is ideal; from a practical and economic standpoint it has several objections.

In the distillation of water the first portion of vapor contains a disproportionate amount of volatile impurities, if such are present. If the distillation is continued to dryness or nearly so the concentrated solution of mineral and organic matters suffers reactions by which more volatile matter is formed and the distillate is again contaminated. For these reasons standard distilled water usually includes only what is technically termed the "middle run of the still," some of the first portion being rejected and the distillation stopped before all the water passes over.

Distilled water, even when obtained with precautions, is not always acceptable for drinking purposes. The taste is flat and suggestive of scorched organic matter. This is often ascribed to the want of aeration, but in many cases the sample is not improved by thorough aeration. Even when so improved, the additional operation adds expense, and unless purified air is used it adds organic matters living and dead. Leffmann believes that the disagreeable taste of distilled water is often due to volatile matters.

The economic production of a high-class distilled water is to be desired both from a sanitary and technical point of view, such as for use by brewers and makers of soft drinks, laundries, paper mills, and many other processes requiring clean and pure water.

Statements are occasionally made that distilled water is too pure and hence not well adapted for drinking purposes, but these statements are not based upon physiological principles or clinical experience.

BOILED WATER

Boiling renders water safe so far as water-borne infections are concerned. It also destroys the toxins and probably renders most poisonous substances of organic origin that may be in the water harmless. Water containing lead and other stable chemical substances injurious to health would not, of course, be rendered safe by boiling.

For the traveler, the camper, and others who must use water of various sources, the character of which cannot be readily ascertained, the only safe procedure is to have his own tea kettle and little alcohol

lamp. Enough water may be boiled in a few minutes in the morning or evening to last twenty-four hours or more for personal use.

Boiling drives off the dissolved gases, which gives to boiled water a flat, mawkish taste. This may be corrected by shaking the water in a bottle or stirring with an egg-beater, or simply exposing it to the air over night, care being taken not to recontaminate it. The disagreeable taste of boiled water is partly due to changes in the organic matter which take place at 100° C. As a matter of fact, it is not necessary to actually boil water to render it safe so far as typhoid, cholera, dysentery, and other non-spore-bearing infections are concerned. A temperature of 60° C. for twenty minutes or 70° C. or 80° C. for a few moments is sufficient. However, in the kitchen, where thermometers and scientific care are not expected, it is better to require the water actually to boil to ensure safety, especially in waters known to be infected or during epidemics. Boiled water may be kept in covered pails or conveniently placed in well stoppered bottles, in which case it may be iced without the risk of contamination.

FILTERS

Slow Sand Filters.—Slow sand filters, also called English filter-beds, consist of large, shallow, tight reservoirs suitably underdrained and containing some five or six feet of stratified filtering material of progressive degrees of fineness, beginning at the bottom with broken stone or gravel and ending with an upper layer of fine sand. The water is passed through such a filter very slowly, from above downward, and the cleansing is done by removing the surface layer of dirty sand.

Slowly passing water in this way through sand purifies it biologically, physically, and chemically; nearly all of the objectionable bacteria as well as other microorganisms are removed and many of the particles in suspension are strained out and much of the organic matter is oxidized.

This process is called "slow" sand filtration to distinguish it from the rapid process known as mechanical filtration. The slow sand filters are spoken of as the English method, or as English filter-beds, because it was in England that they originated; whereas the mechanical filters are spoken of as the American method, because this process was developed in this country to meet our special needs. The student should have a clear comprehension of the differences between these two methods.

The water in the slow sand filter passes very slowly through a layer of sand; the filter chokes by the clogging of the superficial layer of sand, and the cleansing of this type of filter is done by removing this layer or *Schmutzdecke,* as it is called. Mechanical filtration, on the

other hand, consists in first adding a coagulant such as sulphate of alumina and then passing the water rapidly through a layer of sand. The sand is cleansed mechanically by clever devices and by a reversed current of the water.

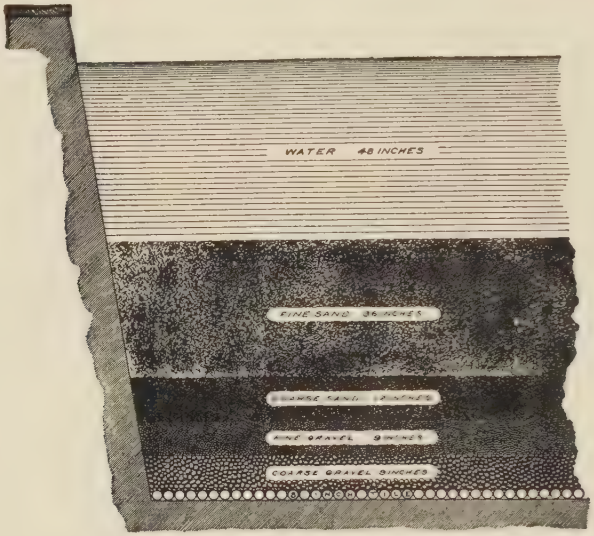


FIG. 106.—SECTION OF AN ENGLISH FILTER BED.

The slow filtration of water through sand originated as an empiric process imitating nature's method of purifying water as it slowly passes through the soil. It was used before the chemistry or bacteriology of the process was understood. In fact, the intimate processes concerned in slow sand filtration are not yet part of our philosophy. We know that the spaces between the sand are enormous when compared with the size of bacteria; nevertheless, over 99 per cent. of the bacteria are held in the superficial layers of the sand. Nitrification and oxidation of organic matter also takes place. The process is not a simple straining, that is, a simple mechanical filtration. It is a "vital" process in which bacterial activity plays a very large part. The bacteria, algæ, and other microorganisms resting upon the upper layer of the sand grow and form a zoogleal mass; each grain of sand becomes coated with a gelatinous and adhesive growth. The layer upon the surface forms a carpet-like mass which constitutes the *Schmutzdecke*. This *Schmutzdecke* effectively holds back the bacteria in the water.

The removal of the bacteria then is largely due to the bacteria, but a visible *Schmutzdecke* is not essential for successful sand filtration. In Hamburg, Lawrence, and other cities a greenish or brownish, slimy *Schmutzdecke* is formed upon the surface of the sand, and gradually becomes so thick and dense as to offer much resistance to the passage

of the water itself. The *Schmutzdecke* is then removed. This can readily be done by scraping or shoveling. Where a visible *Schmutzdecke* is not formed, as in the Washington sand filters, it is probable that the microorganisms which form a zoogleal mass do not find favorable conditions for growth. Nevertheless, in this case the surface layer of the sand becomes clogged in the usual manner and the underlying sand is quite clean. The bacteria that escape the surface action are caught upon and stick to the mucilaginous coating of the sand particles, where they perish as in a trap. The experiments of the Massachusetts Board of Health at Lawrence show that filtration may be as effective from a bacteriological standpoint without the visible *Schmutzdecke* as with it.

Construction and Operation.—In view of the importance of the subject the student should be familiar with the general principles and some of the details concerning the construction and method of operating a slow sand filter.

It is advisable to let the water settle before it is applied to the sand for the reason that this prevents undue choking or clogging of the filters and thus effects a great economy. One of the main items in the cost of maintaining a slow sand filter is the scraping of the surface layer and the washing of the dirty sand. There are other preliminary methods of treating the water before it is applied to the filter. These methods differ with the character of the water, and consist in the main of screening, scrubbing, or coagulation. These processes are discussed more in detail upon another page.

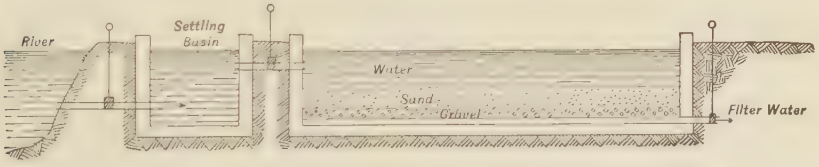


Fig. 107.—The Arrangement of a Slow Sand Filter.

A slow sand filter requires an extensive tract of land, for it should be recalled that only two and one-half to five million gallons of water should be filtered per acre per day. The filter should be conveniently located near the community it is to serve, and the high price of urban property is an important economic consideration. Thus, in Washington it requires 21 acres alone for the filter beds to furnish 63,000,000 gallons of water daily at a 3-million-gallon rate per acre. The settling basins, storage basins for the filtered water, sand-washing apparatus, pumping station, laboratory, and other accessories require considerably more land. The entire filtering surface is divided into units known as filter-beds. The size of each filter-bed has grown with the development of the art. In the filters recently constructed each bed occupies about one acre. Each

bed must be an independent unit, so that the rate of filtration, the cleaning and all other operations may be carried on without disturbing the other beds. The pipes carrying the effluent from each filter-bed must be so arranged that the water may be wasted or utilized. Where the climate is cold, filters should be covered to prevent freezing.

In construction a filter-bed is built very much like an ordinary reinforced concrete reservoir. The bottom and sides must be water-tight, for failure in this regard may be annoying and dangerous for the reason that there may be considerable loss of filtered water or entrance of pollution from the outside if the pressure is reversed. The sides of the bed are usually vertical, although it is some advantage to make them slanting or with horizontal ledges in order to diminish leaks.

The sand may be obtained from a river bed or from sand banks; the grains should be sharp, hard silicates. If the sand contains clay this should be removed by washing before it is used. It is also important that the filtering sand should be free from lime, which has a tendency to make the water hard. The average diameter of the sand best suited usually varies from 0.2 to 0.3 millimeter. It is especially important that the particles should be mainly of the same size. This is determined by establishing the coefficient of uniformity.

The sand used for filtration contains particles of various sizes; the water is forced around the larger particles and through the finer interstices which occupy the intervening spaces, so that it is the finest portion which mainly determines the efficiency of the sand for filtration. According to Hazen, a provisional basis which best accounts for the known facts considers the size of grain such that 10 per cent. by weight of the particles are smaller and 90 per cent. larger than itself. This is considered the *effective size,* and is determined by sifting a weighed amount of the sand through a series of sieves. Another important point in regard to the sand is its degree of uniformity; that is, whether the particles are mainly of the same size or whether there is a great range in their diameters. This is shown by the *uniformity coefficient,* a term used to designate the ratio of the size of grain which is 60 per cent. of the sample finer than itself to the size which is 10 per cent. finer than itself.

The usual thickness of the sand layer varies from 12 to 48 inches. The Imperial Board of Health of Germany has fixed 12 inches as the limit below which the sand should never be scraped. The higher limit is advisable wherever practicable. In this country the usual depth of the sand layer is about 3 feet, and this is reduced by successive scrapings for the purpose of cleaning until it approaches 12 inches, when the sand is replaced. A thick sand layer has a steadying action upon the water on account of the increased friction, and thus aids in preventing irregularities in the rate of filtration.

The sand rests upon a stratified layer of rock and gravel laid in graded sizes which supports it so that it does not work its way down into the underdrains.

The size, position, and nature of the underdrains are a very essential part of the construction of a slow sand filter. The underdrains must be set so that the rate of filtration will be the same in all parts of the filter. If this part of the apparatus is not properly designed in a filter-bed having the broad expanse of an acre the water may pass through the sand in certain portions at the rate of ten or more million gallons while at other portions there may be practically no flow at all.

The depth of the water above the sand is usually 3 feet. In European filters the depth varies from 3 feet to 52 inches. It is comparatively easy through simple mechanical devices to regulate the flow of the applied water so that the depth of the water above the sand will remain uniform.

Probably the most important factor in the operation of a slow sand filter is the rate of filtration. The tendency has been to gradually reduce the rate during the past thirty years. In this country sand filters are usually run at a rate of about 2,500,000 to 3,000,000 gallons per acre per day. Three million gallons is the maximum rate commonly allowed. During times of stress, however, or for other reasons, the rate is sometimes speeded up to five or six million gallons per acre daily. In Hamburg the filters are not allowed to run faster than 1,600,000 gallons, and in Berlin 2,500,000 gallons. Water passed through sand at the rate of 4,800,000 gallons per acre daily has a vertical movement of 3.94 inches in an hour. When the rate is 2,400,000 gallons the vertical motion is 1.97 inches per hour, and when the rate is slower the vertical motion is correspondingly diminished. It will thus be seen that this process is well named in that the water passes very slowly through the filter. This is of fundamental importance because the hour or more during which the water rests upon the surface of the sand and passes through the superficial layer is the critical time when the bacteria are enmeshed in the *Schmutzdecke* or adhere to the particles of sand and the other biological and chemical processes take place. The tendency of engineers is to increase the rate of filtration on account of the evident economy; the tendency of sanitarians is to diminish it so as to keep well within the factors of safety. The rate of filtration may be governed by automatic devices or may be controlled by hand by simply regulating the valve which governs the pipe carrying the effluent from each filter-bed. The friction of the sand layer varies from time to time, so that careful attention is required in order to maintain a steady flow and a constant rate, which is essential, for sudden variations in rate are fatal to the successful purification of water by the slow sand process.

The friction of the sand is measured by the loss of head. The loss of head is the difference between the level of the water above and below the sand layer measured in water gauges. This loss represents the friction or resistance of the sand layer. It greatly increases as the filter clogs up. When a filter is new or perfectly clean the loss of head is usually about 0.2 foot or less; when it exceeds 4 feet the rate of filtration cannot be maintained at 3,000,000 gallons per acre daily with the devices provided, and the filters must be cleaned.

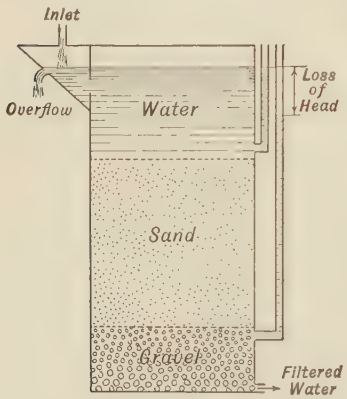


FIG. 108.—DIAGRAM ILLUSTRATING "LOSS OF HEAD."

The length of time a filter may run before the loss of head becomes so great that it becomes unprofitable and requires cleaning varies from a few days to many months. The time depends upon the character of the water, the rate of filtration, and temperature, the formation of the *Schmutzdecke* and many other factors. In cleaning a filter it is sufficient to scrape off only enough sand to a layer that appears clean. As a rule the sand immediately below the surface is not apparently soiled, and usually it is not necessary to take off more than an inch or so of the surface layer. This sand is removed to special cleaning devices, where it is thoroughly washed with filtered water and then stored in bins and replaced when the sand layer reaches a depth of about 12 inches. The *Schmutzdecke* and the surface layers of the sand are usually removed by hand with broad shovels. There are also mechanical devices which accomplish the same purpose. After cleansing, the effluent from a filter-bed should be wasted until the bacteriological examination shows that the filter is again performing efficient work. This may require several days, the time varying with the temperature and other conditions.

EFFICIENCY AND CONTROL OF SLOW SAND FILTERS.—The efficiency of a slow sand filter is mainly measured by a comparison of the number of bacteria in the raw and filtered water. A good filter should eliminate approximately 99 per cent. of the bacteria, provided the applied water is grossly polluted. In any event the filtered water should not contain over 100 bacteria per cubic centimeter and very few colon bacilli. It is to be noted that all the bacteria in the filtered water do not represent those that actually pass through the sand. Some of them grow in the underdrains and gravel layer and are, so far as known, harmless varieties.

In Germany the rate of filtration and other factors are minutely regulated and controlled by official ordinances. In this country the

operation of the filter is left to the individual caprice of the engineer in charge.

A slow sand filter cannot be effectively operated without skilled superintendence of an engineer expert in the art of water purification. It also requires a small laboratory with a competent bacteriologist, who must make daily observations of the applied water and the effluent from each filter. The effluent from a filter not giving good results should be wasted. The water from a new filter, or one just scraped, should not be used until the bacterial results show that it is accomplishing effective purification.

There are many ways in which purer water may be secured, such as the use of lower rates of filtration, finer grained filtering materials, and more complete preliminary treatment, such as settling basins, storage, or chemical coagulation. The filtered water may be further purified with hypochlorite of lime or ozone. It requires a surprisingly small amount of hypochlorite to practically sterilize a filtered water. In Pittsburgh 0.13 part of bleaching powder (measured as available chlorin) per million parts of water is sufficient for this purpose.

Because slow sand filtration has achieved such marked success with some waters and greatly reduced the amount of typhoid is no reason why it should be universally recommended under all circumstances. To recommend slow sand filtration in all cases would be as irrational as to recommend the use of antitoxin in every case of sore throat. A correct diagnosis is essential. Every water cannot be successfully or economically treated by this process alone. Thus, the very turbid waters of our South and West contain particles of clay so fine that they pass a sand filter. No amount of sand filtration will take out some of these particles. The Potomac water in times of high turbidity may be passed through a sand filter three or four times without removing the residual turbidity due to these microscopic particles. To apply a very turbid water to a sand filter soon chokes it and adds unnecessarily to the difficulty and expense of the process. The particles may be so fine that they will not all settle even when the water is given long storage. There are several ways of solving this problem, which is of first magnitude for the purification of the surface water of a large part of our country. One of the best ways is to provide large storage reservoirs, so that the water may be taken from the river only at favorable times, rejecting the flow during periods of high turbidity. Another is to use preliminary coagulation with aluminium sulphate and provide for sedimentation before applying the water to the sand. Much of the turbidity may be removed by a rapid preliminary filtration through some coarse material such as charcoal, sponge, etc. This process is known as scrubbing. No general rule can be set down. Waters differ radically, and the same stream varies from time to time. Each problem must be

studied and solved in relation to its own special condition. Whether the filtered water should be further purified with bleaching powder or ozone depends upon circumstances.

Results of Slow Sand Filtration.—The good results of purifying water by the slow sand method have been abundantly demonstrated in Altoona, near Hamburg, in 1892, during the cholera epidemic; also in Hamburg since 1893 and in Lawrence also since 1893; further in Albany, Philadelphia, Pittsburgh, Berlin, Paris, and many English cities. It should be noted especially at Albany that the typhoid rate did not come down immediately after filtration. It sometimes requires one or two years to reach the residual or "normal" rate. In a few instances, such as Washington, D. C., and Youngstown, O., filtration of the water was not followed by a noticeable diminution in the typhoid rates.

The following American cities purify their water supply by slow sand filtration:

Sand Filters

	Population, 1900	Capacity of Filters in Gallons per Day
Philadelphia, Penn	1,293,697	420,000,000
Pittsburgh, Penn	321,616	100,000,000
Washington, D. C	278,718	87,000,000
Providence, R. I	175,597	24,000,000
Indianapolis, Ind	169,164	24,000,000
Denver, Colo	133,859	30,000,000
New Haven, Conn. (in part)	108,027	15,000,000
Albany, N. Y	94,151	17,000,000
Reading, Penn. (in part)	78,961	5,750,000
Lawrence, Mass	62,559	5,000,000
Yonkers, N. Y. (in part)	47,931	7,500,000
Superior, Wis	31,091	5,000,000
Poughkeepsie, N. Y	24,029	3,000,000

And fully 25 smaller places.

The best results in water purification, as measured by the improvement in the health and reduction of the death rate among those who use the water, have been obtained with slow sand filters. Hazen believes that this is probably because the method is an old one, has been long and carefully studied, and has been applied on a large scale in well-perfected forms for many years, rather than to any natural superiority of the method.

The purification of water through slow sand filtration not only diminishes the amount of typhoid and other water-borne intestinal infections, but is believed also to reduce the general death rate. This fact, known as the Mills-Reinecke phenomenon, is discussed on page 804.

Mechanical Filters.—The essential and characteristic features of

mechanical filtration are: (1) The addition of a chemical precipitant or coagulant to the water, and (2) then passing the water rapidly through a layer of sand. The filtering sand is contained in a large wooden, iron, or concrete tank so arranged that it can be mechanically washed.[1] These filters are well named, not only because the filtering sand is washed mechanically, but because the action is more strictly a mechanical straining, whereas biological processes are the main features in the purification of water passing through a slow sand filter. In the Hyatt and Jewell filters the sand is agitated by a revolving rake as the reversed flow of water washes the sand. In the Continental type the sand is agitated by compressed air.

The coagulants commonly used are sulphate of aluminium, sometimes alum, occasionally sulphate of iron. The alkaline carbonates present in the water decompose the aluminium sulphate with the formation of aluminium hydrate, which is thrown out of solution as a flocculent, colloidal, jelly-like precipitate. The reaction is as follows:

$$Al_2(SO_4)_3 + 3Ca(CO_3) = Al_2(CO_3)_3 + 3CaSO_4$$
$$Al_2(CO_3)_3 + 6H_2O = Al_2(OH)_3 + 3H_2O + 3CO_2$$

The calcium carbonate is necessary to break up the alum, and if not normally present in the water some lime or soda must be added. The precipitated aluminium hydrate clears the water very much as white of egg clears coffee. Suspended matter, including bacteria and inorganic particles, are enmeshed and deposited on the surface of the sand. When this deposit becomes abundant enough to clog the filter the filter is washed by reversing the flow and mechanically agitating the sand. Thus it will be seen that an artificially inorganic *Schmutzdecke* is produced upon mechanical filters instead of the natural organic *Schmutzdecke* of the slow sand filter-bed.

It is advisable to provide coagulating basins to hold the water for a short time after it has received the coagulant, in order to allow the chemical reaction resulting from the treatment to take place. Such basins also serve to remove by sedimentation the greater part of the precipitate, and the filters therefore do not clog so readily, and cleansing is not required so frequently.

The rate at which water is passed through mechanical filters is very great when compared with slow sand filters. Rates varying from 100,000,000 to 150,000,000 gallons per acre per day are used.

One hundred and twenty-five million gallons per acre daily may be

[1] Mechanical filters date from 1884, when the process was patented by J. W. Hyatt and Professor Albert R. Leeds. The Hyatt patent expired in 1901, and since then numerous improvements in details have been made and patented, considerably improving the art of cleaning water through this process.

taken as a fair average of what is to be expected of them. On account of the rapid rate of filtration there is great economy of space. However, while the mechanical filters are cheaper when first cost is considered, the advantage is with slow sand filters as far as cost of maintenance is concerned.

The proper amount of coagulant is added to the water by means of a small automatic measuring apparatus. It requires, as a rule, about one or two grains of alum or sulphate of aluminium for each gallon of water to be treated. The amount of alum added to the water must vary from time to time, depending upon the turbidity, the reaction, and also upon the amount of calcium carbonate in the water. The turbidity and composition of many of our streams vary suddenly and require a watchful eye. If too little alum is added the effluent will not be clear; if too much is used the effluent will contain the excess of alum in solution. Mechanical filters, therefore, require intelligent and constant supervision in order to furnish satisfactory results.

Mechanical filtration meets with special favor in this country because it affords a comparatively cheap method of supplying a clean-looking water from a very turbid source. The process is particularly applicable to the muddy streams of our South and West. In fact, it is the only known method of rendering some of these waters quite free of turbidity.

Mechanical filters, when properly manipulated, will take out from 95 to 99 per cent. of the bacteria contained in the raw water. The bacterial purification, however, is not as constant and uniformly high as that obtained by slow sand filtration. The aluminium hydrate also takes out much of the soluble coloring matter which the water may contain, as well as its turbidity.

Judged by the effects upon morbidity and mortality, mechanical filtration of water has in no instance given the same satisfactory results afforded by slow sand filtration. Most of the mechanical filters in use in America have fallen far short in hygienic efficiency. This was especially true with the old and inferior plants which often were without skilful supervision. According to Hazen, the mechanical filters that have taken advantage of all the improvements in construction and that are operated with skill and experienced superintendence are doing as good work, measured by bacterial efficiency, as the corresponding slow sand filters, and Hazen believes that in time the hygienic efficiency will show corresponding results from them. The sanitarian, however, is compelled to regard the mechanical filtration of water as still in the experimental stage.

THE DIFFERENCE BETWEEN SLOW SAND AND MECHANICAL FILTRATION

Slow Sand Filtration	*Mechanical Filtration*
English system or English filter-beds —originated in England.	American system — developed in America to meet our special needs.
Has been long in use and effectiveness is established.	Comparatively recent (since 1884), and effectiveness not yet established.
Preliminary treatment not an essential part of the process, though sometimes desirable.	A coagulant is first added to the water—sulphate of aluminium, alum, or sulphate of iron.
Water passes slowly through a layer of sand, in large, shallow, tight reservoirs.	Water passes rapidly through a layer of sand in small wooden, concrete, or iron tanks.
Usual rates from 1,600,000 to 5,000,000 gallons per acre per day.	Usual rates 100 to 200 times as rapid —100,000,000 to 150,000,000 or more gallons per acre daily.
Cleaned by scraping surface layer of sand—*Schmutzdecke*.	Cleaned by reversed flow of water and mechanical agitation of the sand—hence the name "mechanical" filtration.
The process is mainly biological, partly a mechanical straining. Duplicates nature's process of purifying water.	The process is mainly a mechanical straining. An artificial imitation of nature's process.
First cost is large; maintenance comparatively small.	First cost is comparatively small; maintenance large.
Especially serviceable for water having little turbidity.	Especially suitable for turbid waters, containing silt and clay.
Removes about one-third of the coloring matter.	Takes out nearly all of dissolved coloring matter.
Removes about 99 per cent. of the bacteria; action is uniform.	When properly operated removes from 95 to 99 per cent. of bacteria—less uniform.
Favorable effect upon health well established.	Hygienic efficiency not established, but doubtless would be more satisfactory if well operated.

The following is a partial list compiled by Hazen of places in the United States where mechanical filters are at present in use or under construction:

	Population, 1900	Capacity of Filters in Gallons per Day
Cincinnati, Ohio[1]	325,902	112,000,000
New Orleans, La.[1]	287,104	44,000,000
East Jersey Water Company	250,000	32,000,000
Hackensack Water Company	225,000	24,000,000
Louisville, Ky.[1]	204,731	37,500,000
Toledo, Ohio[1]	131,822	20,000,000
Columbus, Ohio[1]	125,560	30,000,000
St. Joseph, Mo	102,979	11,000,000
Atlanta, Ga	89,872	6,000,000
Charleston, S. C	55,807	5,000,000
Kansas City, Kan	51,418	6,500,000
Harrisburg, Penn	50,167	12,000,000
Norfolk, Va	46,624	8,000,000
Youngstown, Ohio	44,885	10,000,000
Binghamton, N. Y.	39,647	8,000,000
Augusta, Ga	39,441	6,000,000
Birmingham, Ala	38,415	.....
Little Rock, Ark	38,307	5,500,000
Terre Haute, Ind	36,673	9,000,000
Dubuque, Iowa	36,297	.....
Quincy, Ill	36,252	4,000,000
Elmira, N. Y.	35,672	7,000,000
Davenport, Iowa	35,254	7,000,000
Chester, Penn	33,988	4,000,000
York, Penn	33,708	4,000,000
Knoxville, Tenn	32,637	4,500,000
Chattanooga, Tenn	30,154	9,000,000
East St. Louis, Ill	29,655	11,000,000
Newcastle, Penn	28,339	4,000,000
Oshkosh, Wis	28,284	2,000,000
Lexington, Ky	26,369	3,500,000
Joplin, Mo	26,023	.....
Cedar Rapids, Iowa	25,656	2,500,000

And fully 125 smaller places.
[1] Building.

Household Filters.—The domestic filter as ordinarily used in the household has limited sanitary value. The purification of water, even by so simple a method as straining, requires a degree of care, knowledge, and experience that is not found in the kitchen. If a water is infected, reliance should not be placed upon any household filter operated in the usual way. It is possible in the laboratory by the use of special precautions to pass water through a Pasteur-Chamberland or a Berkefeld filter so as to obtain a sterile filtrate. This requires skilled bacteriological manipulation of a kind that cannot be attained in ordinary service in the house. I have seen janitors "clean" a filter in such a way as to actually contaminate the water.

There are two main types of household filters: one made of unglazed porcelain (koalin), known as the Pasteur-Chamberland, and the other made of diatomaceous earth, the Berkefeld. Even in the closest grained unglazed porcelain filter the pores of the filter are larger than the bacteria. The bacteria do not get through on account of the tortuous passage: they adhere to the particles that make up the filtering substance. But if conditions are favorable, bacteria, such as typhoid, may soon grow through its walls. The Berkefeld filters of diatomaceous earth are more porous than the Pasteur-Chamberland filters.

When a water is not infected, but turbid, household filters are serviceable in rendering it clear. They are specially useful when the turbidity is due to clay or to iron, or other inorganic particles that may readily be removed by simple straining.

The sanitarian places no reliance upon the filtration of water in the household, and for drinking purposes such water if infected, whether filtered or not, should be boiled. The boiling should be the last process.

Filters of natural stone, charcoal, asbestos, and a great variety of porous substances are on the market for domestic use. These filters may be useful in cleaning water that is turbid, but they cannot be depended upon to purify an infected supply.

Scrubbing or Roughing Filters.—Scrubbers are rapid coarse-grained filters through which turbid water is passed at a very high rate in order to remove coarser particles and some of the turbidity. This process of scrubbing the water is principally used as a preliminary to sand filtration. It is designed to protect the sand filters from clogging up too quickly and thus economize the operation. Scrubbers, also known as roughing filters, consist of some porous substances such as sponge, coke, and lava. The principal difficulty connected with a scrubber is an efficient and economical device for cleaning them, which must be done at frequent intervals.

Screening or straining is used particularly to remove fish and floating leaves, sticks, etc. Screens may consist of steel bars arranged so that they may be easily raked off, or of wire cloth arranged in pairs, so that while one screen is raised for cleaning its mate is below in service. Revolving screens are efficient. The motion should be continuous, and the cleaning is done on that part of the screen above the water by jets of water playing upon it. Screening is of no service in removing algæ or microorganisms, and can only be depended upon to remove the coarse particles, and is only necessary where the water contains such material.

STORAGE

The storage of water is one of the simplest and best means of purifying it. The first cost may be large, but the cost of maintenance is

comparatively trifling. Harmful bacteria soon die in a stored water, the solid particles settle out, the organic matter is largely oxidized, the color is gradually bleached, and other improvements take place. Storage takes advantage of many of nature's methods of purifying water, viz., time, sunlight, dilution, sedimentation, oxidation, and symbiosis.

A stored water may deteriorate in quality owing to the growth of algæ and the decomposition of organic matter. Algæ and diatoms grow in stored water exposed to sunlight, particularly in warm weather. While these organisms are not harmful, they impart disagreeable tastes and odors to the water. (See page 723.) The decomposition of the organic matter in a storage water may also cause unpleasant tastes and odors, especially at the spring and fall overturn. (See page 706.) Waters stored in a closed reservoir keep without deterioration, and the advantage is therefore manifest. Filtered water should always be stored in covered reservoirs, not only to protect it from strong light, but also to prevent contamination from dust and other sources.

SEDIMENTATION

Sedimentation is of limited use in improving the sanitary quality of a water. Sedimentation basins are frequently used as a preliminary process in water purification. It is the cheapest way of removing relatively large particles which will settle out in a moderately short time. There is also a sanitary advantage in that the suspended particles mechanically carry down with them some of the bacteria. The water, as a rule, does not remain in the sedimenting basins long enough to obtain the full effects of storage.

Sedimentation is a very important factor in the bacterial purification of flowing streams. The effect of sedimentation is most manifest when the flow of water is rapid enough to prevent accumulation, at any point, of the products of bacterial multiplication, but not so rapid as to interfere with a comparatively rapid action of gravity. Turbid streams purify themselves through sedimentation more quickly than clear streams, owing to the washing or scouring action of the particles as they fall through the water.

CHEMICAL METHODS OF PURIFYING WATER

Ozone.—Ozone is one of the most satisfactory methods of purifying water from a sanitary standpoint. As a germicide it is the most effective of all the methods used except boiling. A well-ozonized water is practically sterile and the organic matter is partially oxidized. It is true that a few resisting spores are not killed, but these are harmless when

taken by the mouth. The limitations of the ozone process are that it does not clarify the water in any way, and that it has practically no effect upon the mineral salts. From a practical standpoint the expense of producing ozone in sufficient concentration is disproportionately large, but this is an electrical engineering problem which is showing encouraging advance.

As a general rule it is not desirable to add ozone to a dirty or turbid raw water. It is better first to clarify the water by some other method before applying the ozone. The quantity of ozone required for effective bacterial action depends upon the amount of organic impurities contained in the water. Much of the ozone will be used up by these impurities, and this may happen so rapidly that it will not have a chance to act upon the microorganisms.

An impure water containing much organic pollution treated with ozone may give disappointing results, from the fact that unpleasant flavors may be developed. These are doubtless due to the partial oxidation of the decomposing organic matter with the production of nitrogenous compounds not well understood.

For the purification of water ozone is produced by electrical discharges in the atmosphere, and this ozonized air is then brought into intimate contact with the water. To produce the ozone requires a brush discharge. If sparking takes place no ozone is produced. The ozonizing apparatus therefore must be carefully designed, and its operation needs skilled supervision.

A brush discharge is an electrical "effluvium," that is, a bluish-violet glow in the dielectric, which in this case is the air, between the electrodes. For the production of ozone on a large scale the electrodes must be large and placed close together. The electrodes may be made of mica, porcelain, or glass covered with foil, etc.; one of them may be metal. The air passing between the electrodes must be dry, otherwise oxids of nitrogen will form, also peroxid of hydrogen at the expense of the ozone. It is therefore customary to first dry the air by refrigeration or by passing it over unslaked lime before it enters the ozonizer. The temperature of the air in the ozonizer must not go above a certain degree, else ozone will not be formed. The maximum production of ozone takes place at about 25° C. Overheating may be prevented by a water jacket in contact with the electrodes. The voltage must be high —from 8,000 to 20,000 volts; that is, the current must have a small volume, but high potential.

The molecule of ozone (O_3) readily gives up one atom of this gas in a nascent condition. It therefore has a very strong oxidizing action upon organic matter, decolorizes many pigments, especially of vegetable origin, and has a very powerful germicidal action. In this respect the action of ozone corresponds chemically to potassium permanganate, the

hypochlorites, and other powerful oxidizing chemicals used in water purification.

It is necessary to get the ozone out of the water in order to avoid the corrosion of pipes. This may be done by aeration, by means of fountains or cascades. On account of the insolubility of the ozone, it soon disappears. The fact that ozone is largely insoluble in water makes it necessary to bring it into intimate contact with all portions of the water to be treated. This is usually accomplished by allowing the water to trickle downward through tall cylinders filled with coke, lava, or other similar substances while the ozone is admitted to the bottom of the cylinder. The water flows downward, the ozonized air works its way upward, and in that way the desired contact is obtained between the ozone and every portion of the water.

A very small amount of ozone is effective for the purification of water. It only requires a few milligrams per liter. The modern machines produce concentrations as high as 10 grains of ozone per cubic meter of air. The ozone not taken up by the water may be used over and over again. This is accomplished in some of the ozonizing processes by conducting the air that leaves the upper part of the water cylinder back to the ozonizer.

In general, it may be said that, owing to the expense and the electrical and engineering difficulties involved, the ozonizing process is not at present applicable to the purification of water upon a small scale. It has been applied with success upon a large scale in a number of places. The first ozonizing apparatus for the purification of water on a large scale was installed by Siemens-Halske at Lille, France. Other ozonizing plants for purification of drinking water have been installed at Paderborn, Germany; at Ginnekin, for Breda, Holland; at Lille, France; at Wiesbaden, Germany; at Nice, France; Lindsay, Ontario, and other places. At Lindsay the ozone treatment failed because the ozone and the water were not properly mingled. At Wiesbaden much trouble was caused by the oxidation of the iron. Experiments at Ogdensburg, N. Y., failed to remove the color of the water. Where water power may be obtained for the generation of the electricity necessary to produce the ozone the cost is very much lessened. The principal systems at present used for ozonizing water are the Siemens-Halske, the Gerhard, Tindal, De Frise, Otto, Abraham Marmier, Vosmaer, Bridge, Stynis, and others.

Ozone treatment is best adapted to sewage-polluted waters, the appearance of which is satisfactory. Waters of turbid streams are least suited to this treatment. Ozone must now compete with bleaching powder, which has nearly the same effect and is cheaper and simpler. One objection to the treatment of water by ozone is that the electric apparatus is delicate and complicated and requires skilled attendance.

The ozone processes are not yet standardized; at present it is difficult to determine what waters may best be treated with it.

Chlorinated Lime—Bleaching Powder.—Chlorinated lime, popularly known as "chloride of lime" or bleaching powder, also chlorinated soda, has been used for years as a sewage disinfectant. It has recently attracted widespread attention in view of the boldness of the Jersey City Water Company in essaying to comply with its contract to furnish pure water to Jersey City by simply adding a little bleaching powder.

The chief ingredient in chlorinated lime so far as water purification is concerned is the calcium hypochlorite; in chlorinated soda it is the sodium hypochlorite. When bleaching powder is added to water it is in no sense a "chlorin" treatment. The essential action depends solely upon an oxidation quite comparable to the process of ozonization. It is not the oxygen in the hypochlorite, but the oxygen in the water, that is liberated. Thus the calcium hypochlorite combines with the CO_2 to form calcium carbonate, and the chlorin liberated unites with the H of the water to form HCl, thus liberating the oxygen, which accomplishes the destruction of the bacteria. The reaction is expressed as follows:

$$Ca\begin{matrix}OCl\\Cl\end{matrix} + Co_2 = CaCO_3 + Cl_2$$

$$2Cl + H_2O = 2HCl + O$$

The amount of hypochlorites added is always expressed in terms of "available chlorin," although in reality this represents the available oxygen liberated by the chlorin. Thus a good bleaching powder will average 35 per cent. of available chlorin, which is the equivalent of about 7.9 per cent. of available oxygen. By available chlorin is understood the chlorin readily liberated from its combination as determined by the usual thiosulphate titration.

Upon exposure to the air the hypochlorites deteriorate rapidly to the more stable carbonates. Great care must therefore be taken to keep the substance in air-tight containers and to know the correct amount of available chlorin in each lot of the bleach at the time it is used.

The amount of chlorinated lime necessary to add to a water in order to accomplish satisfactory results varies with the composition of the water. Thus Clark and Gage found that 0.1 part of available chlorin per 100,000 effected a satisfactory purification of the Merrimac River water; that is, results were obtained equal to slow sand filtration. *B. coli* was entirely eliminated. They discovered the interesting fact that the hypochlorite is a differential germicide, that it destroys some bacteria more readily than others. When small quantities are employed certain species growing at body temperature are only slightly affected. In Pittsburgh it was found that 0.13 part of chlorinated lime, measured in

terms of available chlorin per 1,000,000 parts of water, was sufficient to practically sterilize the Allegheny River water after it had passed the sand filters. It required as much as 1 part per 1,000,000 to accomplish the same results in the raw water. In Minneapolis from 2 to 4 parts per 1,000,000 have been used. In the Jersey City case, already referred to, 5 pounds of bleaching powder, containing 35 per cent. of available chlorin, are added to each million gallons of the water treated. The raw water in this case is not highly polluted, ranging as low as 30 bacteria per cubic centimeter, and rarely going over 15,000. The number of bacteria in the treated water averages only 15 bacteria per cubic centimeter, and *B. coli* is practically absent. It was found only once out of 455 samples.

Impure waters containing decomposing organic matter or large quantities of organic matter of any kind held in solution may, when attacked by hypochlorites, give rise to unpleasant flavors. These substances appear related to the amins, chloramins, and other substances the exact composition of which requires further study. Hence the chemical sterilization of impure waters without subjecting them to some preliminary treatment may give disappointing results.

Bleaching powder in no sense clarifies a water, and therefore cannot render a turbid supply entirely satisfactory. Its cheapness, reliability, and efficiency, and the ease with which it may be applied make it an attractive method. When added in proper quantities it leaves no undesirable chemical substance in the water. Chlorinated lime has a slight tendency to add a little hardness, while chlorinated soda renders the water more soft. The latter, however, is more expensive than the former. The hypochlorite treatment of water is suitable for the purification of supplies upon a large scale and also for military use, camps, tourists, explorers, and others.

Permanganate of Potash.—Permanganate of potash was much used in India, particularly in wells during cholera epidemics; also in water tanks on board ships, and other places. Enough permanganate is added to secure a faint pink tinge, which indicates a slight excess. The permanganate acts as an oxidizing agent precisely as ozone, or similar to the hypochlorites. It is a powerful germicide, but not sufficiently so in the strength used to depend upon it. If too much is added to wells, springs, etc., so as to kill the fish, frogs, and turtles, the water may be spoiled by putrefaction of their dead bodies. Like all chemical methods, the action is not continuous; the agent expends itself in oxidizing organic matters before attacking the bacteria, and the amount necessary for the purification of a water depends, therefore, upon its character.

Experiments by Clark and Gage show that complete sterilization is not obtained by the use of permanganate of potash. Over 98 per

cent. of the bacteria were eliminated by treating water with 0.5 part to 100,000 in from 4 to 6 hours. Larger amounts of potassium permanganate or longer times gave no better results. The cost of the treatment when using 5 parts per 1,000,000 is from $3 to $4 per million gallons. We therefore see that potassium permanganate has a comparatively low efficiency with a relatively high cost, which will always limit its usefulness. Further, the method is difficult of practical application, being rather slow. Occasionally it may be serviceable on ships, in the field, in an army encampment, or an isolated well.

Alum or Sulphate of Aluminium.—The single and double sulphates of aluminium have long been used to clarify turbid waters. In the amounts used they have no direct germicidal action, nor any direct chemical action upon the water itself. The action is entirely an indirect one, and depends upon the fact that the alkaline carbonates react upon the alum to form aluminium hydrate. This salt has a large colloidal molecule and, being insoluble, is thrown out of solution as a flocculant precipitate which entangles much of the suspended matter and bacteria. In a sense the purification of water with alum corresponds very much to the clearing of coffee with the white of egg. Some of the aluminium hydrate may also combine directly with the organic matter to form undetermined compounds. The reaction is given on page 789. By which it will be seen that if alum is added in just sufficient quantities to a water it leaves no undesirable constituent in the water. This is important, for there is a great prejudice against the addition of a chemical, especially alum, to drinking water. In Washington it is actually forbidden by law, despite the fact that it has been shown that in times of great turbidity the only known method of clearing the Potomac water is by the use of a coagulant such as alum. It has already been pointed out that there are many such turbid waters in our country which contain silt in such fine subdivision that even prolonged sedimentation and repeated filtration will not entirely remove it.

In the use of alum good results depend upon adding it in just the right amount. The quantity will vary with the turbidity and the amount of calcium carbonate contained in the water. This should be carefully determined from time to time, for if not enough alum is added the result is incomplete, and if too much is added it remains in the water as such. The process therefore needs constant supervision, for turbid waters usually come from turbulent streams, which are subject to sudden variations. If the process is left to automatic devices or placed in incompetent hands it is sure to give disappointing results.

Few waters may be satisfactorily purified by the use of alum alone. The alum should be regarded only as one part of the process. Subsequent sedimentation, filtration, or hypochlorite, etc., are necessary, depending upon circumstances. Alum alone should never be depended

upon to purify a sewage-polluted water. When properly combined with filtration it will eliminate a large percentage of the bacteria.

Sulphate of iron and alum in combination are used in many places. At St. Louis it was introduced as an emergency installation to clarify the muddy waters of the Mississippi, to make a good impression during the Louisiana Purchase Exposition in 1904. It gave such satisfactory results that it was decided to continue its use.

Lime and iron are cheaper than sulphate of aluminium. Their application is much more difficult to control adequately, and it should never be undertaken except with the assistance of a competent resident chemist and good appliances for adding the lime in any quantity that may be required by the composition of the water. At St. Louis the water is subject to the iron and lime treatment, followed by subsidence in large basins in which the bulk of the precipitate settles. This partially purified water is then sent to the city without filtration or other treatment.

Metallic Iron: the Anderson Process.—The Anderson process (patented) for the purification of water consists in agitating the water in contact with metallic iron a portion of which is taken into solution as ferrous carbonate. This action is brought about by the CO_2 in the water which attacks the iron. Upon subsequent aeration the ferrous carbonate is oxidized and precipitated out as the insoluble ferric hydrate, which accomplishes all the good and none of the bad effects which follow the use of alum. The precipitate is partially removed by sedimentation, or filtration may complete the process. The process is used on a large scale at Antwerp, Belgium, where the water passes through long revolving cylinders containing baffle plates and loose pieces of metallic iron. As the cylinders revolve the iron is continually carried up and dropped through the water in a constant shower. The water passes slowly from one end of the cylinder to the other.

The process theoretically is an excellent one, but apparently enough iron is not always obtained in solution to accomplish the results when applied on a large scale. Especially when peaty waters are used, it seems impossible to get enough iron into solution in the time which can be allowed; or the inorganic acids may form soluble compounds with the iron, thus defeating the object of the process. Other places where the Anderson process is used are at Dortrecht, Holland, Boulogne-sur-Seine, near Paris, and elsewhere.

Copper Sulphate.—The use of copper sulphate in drinking waters was proposed by George T. Moore of the United States Department of Agriculture in 1904. The original claim was that copper sulphate in minute amounts would poison algæ which produced objectionable tastes and odors, and the further claim was made that it was also capable of destroying typhoid and other pathogenic microorganisms. We know

now that copper sulphate in great dilution is a specific poison for many algæ and other microscopic organisms, but that it has little or no effect upon typhoid, cholera, or dysentery bacilli in the amounts used.

Copper sulphate is used in the proportion of 0.1 to 0.25 part per 1,000,000 parts of water. Some algæ require larger doses. Most of the copper combines with the bodies of the microorganisms and settles with them to the bottom and in this way is removed from the water. If the water is afterwards filtered most of the remaining copper is removed. The copper remaining in the water is in such minute amounts that there seems to be no real danger in using it in this way or even in its occasional use in somewhat larger doses where the water is very bad.

The method of applying the copper is to place weighed quantities of the copper sulphate in loose cloth bags and to tow them back and forth with rowboats through the water of the reservoir until the material is dissolved. It should be remembered that, while the copper kills some species of organisms in the amounts used, it has no effect whatever upon others. In fact, it permits the growth of certain species by removing the retarding symbiants, thus clearing the way for stronger growths of the forms that are not directly affected. Copper sulphate may therefore entirely change the flora in a reservoir. This change is frequently accompanied by a great improvement in odors and tastes. On the other hand, the destruction or suppression of one species may be followed by an overgrowth of an equally objectionable and more hardy form. Therefore the results from the use of copper sulphate for the correction of odors and tastes in water vary from complete successes to utter failure.

It is clearly established that copper sulphate does not prevent or even materially reduce putrefaction and the tastes and odors resulting from it. According to Hazen, the method of treating water with copper sulphate is easily and quickly applied, and considerable good has come from it. The correction is only partial, however, and is not always permanent. It is not therefore to be relied upon in all cases.

ULTRA-VIOLET RAYS

Recently the well-known germicidal power of the ultra-violet rays has been put to practical use in the sterilization of water, milk, and other substances. These rays, of short wave length, may be obtained from the Cooper Hewitt mercury vapor lamp, which is very rich in ultra-violet rays. Nagier conceived the idea that this lamp might be used for the sterilization of water, and the experiments made in France, England, and elsewhere show this assumption to be correct. As glass

is opaque to ultra-violet rays, it is necessary to use quartz or lamps made of fused silica. The apparatus used in the experiments [1] of Thresh and Bealle consists of an aluminium cylinder about 12 inches long by 6 inches in diameter containing a Cooper Hewitt quartz lamp with an internal diaphragm, which causes the water entering at one end to travel along the cylinder in close proximity to the lamp. By an ingenious arrangement the moment the light goes out the flow of water is stopped. This small apparatus is capable of sterilizing 50 to 200 gallons of water per hour, depending upon the character of the water. In clear water many of the bacteria are killed in from 5 to 20 seconds. The resisting spores succumb in 30 to 60 seconds, *B. coli* in 15 to 20 seconds, *B. typhosus* 10 to 20 seconds, cholera vibrio 10 to 15 seconds. The presence of colloidal material or turbidity retards the action of the rays. The current used in these experiments was 6 ampères and 130 volts. The results show that a fairly clear and bright water may be practically sterilized by exposure to ultra-violet rays for a brief time. The simplicity of the apparatus and its comparative cheapness make it attractive, so that it doubtless will receive much attention in the future.

Marseilles recently adopted the ultra-violet rays to purify its water supply. There are preliminary roughing filters, and the water passes the quartz tube mercury arc lamp three times. No *B. coli* were found in the treated water, and the total bacterial reduction was 98.3 per cent. It is probable that the bacteria are killed by exposure to the direct action of the ultra-violet rays themselves. The process does not in any way clarify the water.

Other electrical methods have from time to time been devised for the purification of water, using the water itself as an electrolyte. These processes have not yet been developed to give successful results on a large scale, but much may be hoped from them, and they are worth careful study.

[1] *Lancet*, Dec. 24, 1910.

CHAPTER VI

WATER AND ITS RELATION TO DISEASE

Water is a vehicle for certain infections such as cholera, typhoid fever, dysentery, and other diseases, having their primary seat in the digestive tract. It may carry inorganic poisons such as lead. It is responsible for a large group of nutritional and dietetic disorders less well understood. It may contain qualities which bring about derangements of metabolism resulting in such conditions as goiter; further, it may be the medium for carrying infections now not generally regarded as water-borne, or it may lower resistance so as to favor infections not water-borne. It is also occasionally responsible for conveying animal parasites, amebæ, worms, etc.

While water has an established place among the carriers of certain infections, it has not a supreme or exclusive place, and this should be kept carefully before us. The tendency to exaggerate the importance of water as a bearer of disease and death has sometimes led to overstatement. The facts are bad enough and do not require extravagant language to emphasize their importance. The greatest danger in water is pollution from human sources. All the discharges from the body: urine, feces, expectoration, secretions from the nose, and washings from the skin, find their way sooner or later into our streams, especially where modern water-carriage systems are installed for the disposal of wastes. All sewage-polluted water must be regarded as dangerous, whether there are any known cases of typhoid fever on the watershed or not. It is highly probable that the sewage of large communities always contains typhoid bacilli in larger or smaller numbers, because in large communities typhoid fever does not die out completely at any time, and carriers and missed cases are growing in interest and importance.

Water differs in several essential particulars from any other article of diet. Above all, it is partaken of raw, while perhaps 90 per cent. of all our other food is disinfected by cooking before it is used. Again, it is a vehicle which comes in contact with many objects spread over broad acres, and it is the natural vehicle for the removal of wastes from these areas. Its great solvent and erosive powers favor this action.

The relation of water supply to sickness and death has been shown with force in many cities, notably at Lowell and Lawrence, Mass.; in Albany, N. Y.; at Jersey City and Newark, N. J.; at Philadelphia and Pittsburgh, Pa.; at Chicago, Ill.; and abroad at London, Paris, Hamburg, Altona, Berlin, and many other cities.

THE MILLS-REINCKE PHENOMENON

Following the filtration of the water supply of Lawrence, Mass., in September, 1893, Mr. Hiram F. Mills, a member of the State Board of Health of Massachusetts, noted that a marked decrease in the general death rate of the city, and not merely in the death rate from typhoid fever, was taking place. About the same time (May, 1893) filtered Elbe River water was furnished the city of Hamburg, and Dr. J. J. Reincke, health officer of that city, in his successive annual reports, noticed that the general death rate was declining more rapidly than could possibly be accounted for by the deaths from typhoid fever alone. To this important discovery Sedgwick and MacNutt have given the name of the "Mills-Reincke phenomenon."[1] In 1904 Mr. Allen Hazen, a sanitary engineer, formulated a numerical expression for the comparative effect of water purification upon typhoid fever mortality and total mortality. He said that, "where one death from typhoid fever has been avoided by the use of a better water, a certain number of deaths, probably two or three, from other causes have been avoided." The Mills-Reincke phenomenon and Hazen's theorem have been searchingly studied by Sedgwick and MacNutt, and the student is advised to read the original article referred to in the footnote. These authorities examined the vital statistics of the cities of Lawrence, Mass., and Hamburg, Germany, and also of Lowell, Mass., Albany, Binghamton, and Watertown, N. Y. They found abundant evidence of the great life-saving power of a purified water in preventing many diseases other than typhoid fever in the cities studied, except Watertown, and in this case it is possible that the purification of the public water supply has been as yet relatively imperfect. It is further to be noted that the method of purification used at Watertown is mechanical filtration.

Sedgwick and MacNutt express the opinion that Mr. Hazen's theorem applied to the cities they studied, with the exception of Watertown, appears to be sound and conservative. In Hamburg the saving in typhoid mortality was slight in comparison with the saving of mortality in other diseases combined; that is, roughly, only about

[1] W. T. Sedgwick and J. S. MacNutt: "The Mills-Reincke Phenomenon and Hazen's Theorem Concerning the Decrease of Mortality from Diseases Other Than Typhoid Fever Following the Purification of Public Water Supplies," *Jour. Infect. Dis.*, Vol. VII, No. 4, Aug. 24, 1910, pp. 489-564.

1 to 16. In the other cities the ratios differed widely from this. Thus, at Lawrence it was 1 to 4.4, at Lowell 1 to 6.0, in Albany about 1 to 4.1, and in Binghamton only about 1 to 1.5. It is clear, therefore, that Hazen's theorem is merely a convenient formula rather than a precise methematical expression.

One of the most surprising results of these studies is the disclosure of the remarkable relation subsisting between polluted water and infant mortality. This was emphasized especially by Dr. Reincke at Hamburg. Closely associated with infant mortality stand diarrhea and gastro-intestinal disorders in relation to polluted water, which now bids fair to assume a causal importance in these diseases second only to that of contaminated milk.

In regard to tuberculosis the evidence, though less striking, is interesting and suggestive. Sedgwick and MacNutt state that, "inasmuch as they have been unable even after the most careful investigation to discover any other possible explanation of the figures, they are forced to the conclusion that a considerable portion of the decline in mortality from tuberculosis in Lawrence and Lowell during the years immediately following a change from a polluted water supply was due to that change, and in line with this conclusion a similar explanation appears more than probable for Hamburg. A somewhat similar relation stands for pneumonia, bronchitis, and the acute respiratory diseases."

The question naturally arises as to what such decline of mortality observed in the Mills-Reincke phenomenon for diseases other than typhoid fever is due. The natural suggestion is that it either results from an increased vital resistance resulting from the use of purer water or an exclusion of the disease germs, or perhaps the phenomenon might be due to a combination and coöperation of these two factors.

McLaughlin[1] has also stated the relation of a sewage-polluted water to infant mortality, and concludes that it is certain that in practically every instance, in addition to a lessened number of deaths from typhoid fever, the substitution of a safe for a polluted water supply results in the saving of many lives from diseases which are not reported as typhoid fever. Hazen's theorem has also been studied by Arthur Lederer,[2] who finds a large number of affirmative statistical results from which, together with our direct and indirect proof of the prevalence of water-borne diseases, it seems safe to assume that the influence of an improved water supply upon the death rate in general is correct. The theorem seems well borne out by the figures in the following table:

[1] *Pub. Health Reports*, Vol. XXVII, No. 17, Apr. 26, 1912.
[2] Arthur Lederer: *Amer. Journal Public Hygiene.* June, 1910, p. 304.

City	General Death Rate Before Change of Water Supply	Same After	Percentage Reduction	Typhoid Fever Death Rate Before Change of Supply	Same After	Percentage Reduction
Providence, R. I.	19.3	19.0	+ 1.6	21.8	13.7	+37.2
St. Louis, Mo.	18.0	16.1	+10.6	39.2	19.1	+51.3
Youngstown, O.	15.6	15.1	+ 3.2	96.1	39.1	+59.4
Ithaca, N. Y.	16.4	15.1	+ 7.9	108.8	25.3	+76.8
Paducah, Ky.	23.4	17.8	+23.9	82.1	78.7	+ 4.2
Watertown, N. Y.	15.5	17.2	—11.1	100.6	38.2	+62.1
Paterson, N. J.	17.2	16.5	+ 4.1	28.2	11.9	+57.8
Binghamton, N. Y.	17.6	17.6	0	40.8	13.4	+67.2
Average	17.8	16.8	+ 5.7	64.7	29.9	+53.8

NON-SPECIFIC DISEASES DUE TO WATER

Impure water is responsible for disorders other than the specific gastrointestinal infections, but these disorders are often obscure or overlooked. It is not always plain just what quality or what impurity in the water is responsible for these non-specific disorders, and the diseases themselves may present a vague and ill-defined clinical picture. The relationship has been worked out in only a few instances.

A turbid or malodorous water may not in itself be particularly injurious to health, but, on account of its unattractive appearance or repulsive condition, less may be taken than is necessary for the maintenance of good health. In this way water may be indirectly responsible for much harm. The drinking of too little water is a very common dietetic error.

While a polluted water may not carry specific germs, it may so undermine health or lower resistance as to favor infections not usually associated with the digestive tract, such as pneumonia and tuberculosis and the diseases responsible for infant mortality.

From the nature of the case the effects of an impure water cannot always be measured by gross results, but the cumulative or separate action of small effects often repeated may result in deranged digestion, altered metabolism, irritation of delicate membranes or sensitive organs and structures, which may lead to or hasten the course of chronic diseases.

The *organic matter* in the quantities usually contained in a natural water is not of itself harmful. This organic matter, however, does not stay in its native state, but soon putrefies, and it is suspected that some of the intermediate products of putrefaction may have toxic potency. Ordinarily these toxic substances are in minute quantities, or at least

in great dilution, but under certain circumstances they may accumulate in noticeable concentration. Further, while persons habitually taking such toxic substances may soon become immune, the new-comer will not be so fortunate. The case of organic matter in water is not a clear one, and sanitarians have ever erred on the safe side in condemning waters containing much organic matter. It is well known that if the organic matter is not derived from sewage it is probably harmless. Thus, in the case of organic matter of vegetable origin, Mason has been able to find but few cases of illness traceable to peaty waters. In such instances the patients suffered from a mild and transient form of diarrhea. I am familiar with an outbreak of diarrhea traced to a dead fish caught in the water meter of a hospital. This is probably a type of water-borne disease due to organic pollution which is not infrequent. Whether in such cases the trouble is due to bacteria or to bacterial toxins, or to the degradation products of protein decomposition, cannot always be made out.

As far as the *inorganic impurities* usually found in water are concerned, the chlorids, carbonates, sulphates, and silicates, and lime, magnesia, and aluminium can scarcely be harmful in the amounts ordinarily found. It is commonly stated that water containing 500 parts per million, or 30 grains per gallon, of clay and silt is unfit for drinking purposes, on account of its irritating effects upon the gastrointestinal tract; but beyond this probability, turbidity is of no special sanitary significance, unless the water also contains metallic poisons or objectionable chemicals.

An attempt has frequently been made to correlate the formation of concretions such as urinary and biliary calculi with the inorganic salts in water. We now know that biliary calculi usually form about a colon bacillus or a typhoid bacillus or about some pathological particle as a nucleus, and that urinary calculi probably have a similar pathogenesis. There is no known relation between these concretions in the body and the inorganic salts in water, even those in a very hard water. The relation of inorganic substances in water to goiter will be discussed separately.

Goiter.—Goiter or struma is a chronic enlargement of the thyroid gland. It occurs as epidemics, is endemic in places, and sporadic cases may arise anywhere. Goiter has many of the earmarks of an infectious disease, although it is not communicable from person to person, the cause being derived from his environment. The epidemics are usually of short duration, limited extent, and commonly occur in goiter regions.

The classic home of endemic goiter is in the Swiss Alps. In certain regions of these mountains it is very prevalent. Thus, in Piedmont it sometimes affects more than two out of every three of the inhabitants. It also occurs in the mountains of Austria, France, and

Germany, and there are a few endemic centers in Norway, Sweden, Finland, and the Baltic provinces. The traditional seat of goiter in England is in Derbyshire, while Sussex and Hampshire have also been affected. There are many endemic centers in the mountains of Asia, Japan, the Asiatic Islands, Africa, Mexico, and South America. The early explorers found it among the North American Indians, as Munsen has in more recent times in the Eskimos. According to Osler, Dock, and Adami, the region of our Great Lakes shows considerable numbers, but in the United States and Canada the goiters are not large and cretinism is rare. The absolute number of goiter subjects in countries with endemics of severe degree is of great social and economic importance. In France, Mayet (1900) estimates the number at 400,000. The drain on the country is better expressed by the number of cretins. In Cisleithan, Austria, there were in 1883 a total of 12,815, or 71 per 100,000; in one district in Styria a proportion of 1,045 in 100,000. In Piedmont, Lombardy, and Venetia there were in 1883 12,882 cretins in a population of 9,565,038 (Dock). It will therefore be seen that this disease, which is undoubtedly preventable, but the cause of which has not yet been satisfactorily unraveled, deserves careful study.

Goiter is a disease which is caused by some poison or possibly infection taken into the system with the water or perhaps some other article of diet. There is much evidence that drinking water is responsible; also some that it is not. There is no doubt that remarkably good effects have been obtained in Switzerland and Italy by the introduction of good drinking water. For a long time glacial waters were believed to be responsible, but this view has now been abandoned as the cause of goiter. Suspicion has fallen upon certain inorganic constituents of water, but all these studies have resulted negatively. Thus the magnesium limestone, iron, and iodin have each in turn been accused. It is known that goiter may occur where water is hard or soft, or in water with or without iron. The relation of iodin in water to goiter is an attractive theory. It is known that iodin under certain circumstances stimulates the function of the thyroid gland and produces the train of symptoms associated with exophthalmic goiter. As goiters occur in regions in which the water does not contain unusual amounts of iodin, this relationship is therefore doubtful.

It is assumed that some constituent in the water is responsible for goiter from the well-known fact that there are goiter wells in France and Switzerland. These waters are used successfully for the intentional production of the disease with the view of escaping compulsory military service.

The relation of water to goiter is also illustrated in Vienna. This city long boasted of the best water among all European cities. It is brought in long aqueducts and subterranean pipes from the Schneeberg,

a mountain group about 6,000 feet high and 85 miles to the north of the city. This water, used since 1872, put a stop to typhoid and other gastrointestinal diseases. The water comes from limestone formations, and has a low degree of hardness, owing to the absence of vegetation upon the catchment area. Since 1873 the number of goiters in Vienna have increased 200 per cent, and popular belief always pointed to the water as the cause. The water used by the inhabitants in many of the goiter regions in Switzerland comes from similar limestone formations.

It has recently been shown that "goiter" is very common in trout in certain regions. Thus, in our own endemic area about the Great Lakes many trout have enlarged thyroid glands. In trout hatcheries almost all the fish may suffer from goiters, some very large, provided three conditions are present. These are: (1) overcrowding; (2) overfeeding; and (3) pollution of the water. It is stated that in the absence of any one of these three conditions the thyroid glands do not enlarge. These enlarged glands have been described by Gaylord as cancers.

For the reason that no definite correlation has been demonstrated between any inorganic impurity in a water and the occurrence of goiter, investigators such as Kocher, Ewald, Bircher, and others are inclined to think that a microorganism is the cause of the trouble. This theory would explain the definite endemic distribution of the disease.

Bircher[1] has shown that goiter occurs essentially upon marine deposits of paleozoic, triassic, and tertiary periods, whereas volcanic formations, crystalline rock of archaic age, stratified deposits of the Jura and Kreidemeer, and all fresh-water deposits are free. These facts led Wilms[2] to assume that the cause of goiter is not a living organism, but that it is due to unknown substances derived from the bodies of marine animals. He speculates that these substances may be toxalbumins or ferments. Experiments upon rats show that water from a goiter well in Basel produces hypertrophy of the thyroid. This, however, does not occur if the water is heated above 80° C. The hypertrophy takes place in the rats who drink this water which has been passed through a Berkefeld filter. The water looks clear and fine, has a moderate lime content, and is slightly high in ammonia.

Lobenhoffer[3] studied the presence of goiter in the Unterfranken district. In some of the town 21 to 26 per cent. of the inhabitants are affected. The endemic regions correspond to certain geological formations from which the drinking water is derived. Water from shell limestone is the main goiter producer, but in a milder degree red sandstone and other formations are involved. Boiling the water seems to do

[1] *Med. Klinik*, 1908, Heft, 6.
[2] *Deutsche med. Wochenschr.*, March 31, 1910, p. 604.
[3] Lobenhoffer, W.: "Die Verbreitung des Kropfes in Unterfranken," *Mitteilung aus den Grenzgebieten der Med. und Chir.*, Jena, XXIV, No. 3, pp. 383-606.

away with its goiter-producing properties. Lobenhoffer believes that the latest researches in regard to the causal agent of goiter seem to demonstrate that it is a purely chemical substratum substance which enters the water as a toxin, but which is certainly destroyed at 70° C. He also believes that it is possible that filtering through certain substances or treating with ultra-violet rays may have the same effect.

The prevention of goiter therefore consists in the elimination of factors that are known or suspected of being able to produce the disease, such as water from certain sources. If possible, persons in predisposed families should leave goiter districts and live in healthy localities. The drinking water should be boiled, for experimental evidence demonstrates that the "poison," whatever it may be, in water is destroyed by boiling. Filtration is not sufficient, for experiments have shown that it will pass a Berkefeld filter. Tight collars and occupations that induce congestion of the head should be avoided. The satisfactory control of the disease must await further studies into its causation. In the meantime improved sanitation in its broadest sense would doubtless diminish the incidence to this disease.

Lead Poisoning.—Lead is practically never found in natural waters. The source of the lead in the water is almost always lead service pipes, or some other lead object used in collecting, storing, or delivering the water. Lead is the most dangerous inorganic substance with which our drinking water is ordinarily contaminated. Lead poisoning from this source is much more common than it is given credit for. A celebrated instance of lead poisoning occurred in Lancashire and Yorkshire, England. The water came from peaty moorlands and was delivered through lead pipes. The citizens of these towns experienced a mysterious bodily derangement for some years, until it was finally discovered that lead poisoning was prevalent. In many other places, as Somerfeld, Germany, and Lowell, Mass., numerous cases of lead poisoning due to the action of water in lead pipes have been reported.

Enormous quantities of lead service pipes are still in use, not only in the old plumbing, but in the newer installations. It is so pliable that plumbers find it much easier to bend it around corners and angles than to make the usual connections with iron or brass pipe, and it is therefore a great temptation to put in short lengths of it in difficult places. Lead poisoning may, under certain circumstances, come from a few feet of lead pipe. The various factors that determine the corrosive action of water upon lead are very complex. It is not possible to determine by chemical tests whether or not a water has plumbo-solvent action. All natural waters have some solvent power. The only sure method of determining to what degree a given water will take up lead is by testing the question experimentally under practical conditions and establishing the amount of lead taken up.

The way by which water takes up lead is first through the formation of lead oxid. This oxidation is favored by the amount of oxygen carried in the water, possibly aided by the nitrates and nitrites serving as oxygen carriers. The lead oxid may then be dissolved, more rapidly if the water is acid, or may be washed away by the currents in the state of a fine powder in suspension.

As a general rule clean (pure) waters have a greater corrosive action upon lead than turbid waters. This is partly for the reason that the mud coats the pipes and protects them mechanically. Acid waters are almost sure to take up lead if allowed to come in contact with that metal. Even so feeble an acid as carbonic acid may under certain circumstances greatly increase the plumbo-solvent action of water. Soda water (highly charged with CO_2 under pressure) takes up relatively large quantities; as if lead pipes are used in soda water fountains or "syphon" bottles. Waters containing carbonates or sulphates are not apt to take up lead because the corresponding salts of lead are insoluble, and thus form a protecting coating. Even though a water has no plumbo-solvent action, the use of lead piping, lead cooking utensils, lead-lined cisterns, etc., is entirely unjustified for domestic service, for the reason that under certain circumstances electrolytic action, changes in the character of the water, or other causes may lead to lead poisoning.

Various conditions affect the plumbo-solvent action of water, such as the duration of contact, the temperature, the pressure, the season of the year, the purity of the lead, etc. Water remaining in the pipes all night naturally takes up more lead than the water that flows more or less rapidly during the day. Lead pipes were formerly used in soda water fountains and the employee who took the first drink in the morning before the proprietor arrived received a concentrated dose. Hot water has a greater solvent action than cold water; so, also, increase in pressure up to 140 pounds to the square inch. For some unexplained reason more lead is often found in the water during the winter than during the summer. The purer the lead in the pipes the freer the solvent action. New pipes give up more lead than old pipes. However, in some cases the poisoning manifested itself only after the pipe had been in use for years. Lead pipes are purer now than formerly, owing to profitable methods of extracting the silver and other metals with which it is frequently associated. If the lead is combined with copper, zinc, or tin the lead passes into the water more quickly in consequence of galvanic action than when pure lead is used. Electrolytic action favors the solution of lead, and the modern method of grounding electric currents adds to the danger.

The various conditions of water that favor plumbo-solvent action are: Those containing free acid, such as soft, peaty waters; those containing much oxygen and little dissolved salts, that is, soft waters, such as

rain water; those containing organic matter, nitrites, and nitrates, that is, sewage-contaminated water in the stage of oxidation; those containing chlorids, because chlorids dissolve the protecting film of carbonates. Waters that act least upon lead are turbid waters and hard waters, especially those containing free CO_2, for here again carbonates are formed which protect the water with an insoluble film. However, if CO_2 is present in excess or under pressure the carbonates are redissolved.

It will therefore be seen that the purest, softest, and best aerated waters are especially prone to act upon lead. Distilled water will take up lead even from impure zinc pipes (containing some lead) used on board ships. Absolutely pure water probably has no appreciable action upon metals such as lead, iron, and zinc, but absolutely pure waters are not found in nature. The plumbo-solvent action is in part a mechanical erosion, in part a chemical solution, and in part results from electrolytic action.

Symptoms.—The symptoms of lead poisoning are sometimes vague and readily overlooked. Fatal poisoning may be caused when very little lead is taken with the water each day; the action is cumulative and the course of the intoxication is chronic; the immediate and remote effects are serious.

The usual symptoms of chronic lead poisoning are anemia, dyspepsia, depression, constipation, colic; various forms of paralysis, especially paralysis of the extensor muscles of the forearm leading to wrist-drop; a blue line along the edges of the gums, due to the formation of sulphid of lead deposited in the tissues. Optic neuritis may come on. There is an increase in the blood pressure. Chronic lead poisoning leads to arteriosclerosis, fibrosis of the kidneys, and the remote consequences of these changes. Muscular paresis, pain and swelling of the joints, often occur and may be mistaken for "rheumatism." In some cases gout is closely simulated. The pain is usually worse at night.

The individual susceptibility to lead poisoning varies remarkably. Of a number of individuals equally exposed some will suffer and others escape. Of those who suffer the degree of intoxication varies considerably. It is quite common to find that among the members of a family using a water containing lead only one is stricken, while the others seem to be immune; that is, they either do not absorb the lead or are able to eliminate it.

Mild cases of lead poisoning may show only symptoms of anemia, vague or fugitive pains, or a mild type of peripheral neuritis. This stage of lead poisoning, which does not vary essentially from other intoxications of mild degree, is readily overlooked clinically.

Lead is absorbed from the intestines and eliminated by the kidneys and the liver. It therefore may appear in either the urine or feces. Lead in the urine is always associated with albumin, and may be inter-

mittent. That is, a well-marked case of lead poisoning may excrete urine free from both lead and albumin. However, if the feces are examined they will be found to contain lead.

Lead poisoning may occur when a comparatively small surface of lead is exposed to the solvent action of the water. This is well illustrated in the following cases:[1]

Case 1.—A man about fifty years old contracted lead poisoning from using cistern water. Twelve feet of the service pipe was lead, and almost wholly in the water, as it was bent at right angles and ran across the cistern under the water.

Case 2.—Mrs. W., sixty-six years of age, contracted lead poisoning from a well water which was contaminated from an old lead clock weight which had been accidentally dropped into the well. The clock weight had been in the water about fourteen months before the appearance of symptoms. The well was pumped free of water and the clock weight found and removed. In two weeks from this time Mrs. W. noticed an improvement in her lameness, and in four months she was entirely well.

Case 3.—In this case the patient was poisoned by cistern water pumped through 10 feet of lead pipe. The symptoms were acute multiple peripheral neuritis, with extensive paralysis. After the lead in the water was removed recovery was only partial after a period of two years.

SPECIFIC DISEASES DUE TO WATER

The principal diseases of man contracted by drinking infected water are typhoid fever, cholera, and dysentery. Water-borne epidemics of these diseases have frequently occurred in the history of the world. It should be remembered that endemic and sporadic cases may also contract their infections through water. The great water-borne tragedies have for a time occupied an exaggerated position. They overshadowed the less dramatic, but more insidious, and nevertheless frequent modes of transmission of infection through other channels, especially "contacts." It is only in recent years, since the water supplies of most of our large communities have been very much improved, so that water-borne epidemics have been excluded, that sanitarians have appreciated the quantitative rôle played by water as a medium of convection in distributing pathogenic microorganisms.

It is worthy of note that almost all the large water-borne outbreaks that have been investigated have been traced to a quick transfer of the infected material from the patient to the victim. The greater the distance and the longer the time between the source of the infection

[1] *Bull. State Board of Health*, Maine, Jan., 1909, Vol. I, No. 21.

and the use of the water, the less are the chances of harm. This we now understand as the result of several factors which have been discussed.

It is doubtful whether typhoid, cholera, or dysentery bacilli multiply in water under natural conditions, certainly to no great extent. Almost all the great water-borne epidemics of typhoid fever occur in the spring, winter, or fall of the year, when the water is very cold. Water-borne epidemics of typhoid in the summertime, when the conditions seem favorable for multiplication of the bacilli, are relatively infrequent. Assuming that in the case of typhoid there is no multiplication of the bacilli in the water, the dilution must have been enormous in many of the cases recorded; that is, there must have been very few typhoid bacilli in a tumblerful of water. If these facts are correct it illustrates how very few bacteria, when fresh and virulent, may induce disease. The experimental data from the laboratory indicate that the healthy organism may, as a rule, successfully overcome small doses of infection. Feeding experiments, especially upon the lower animals, under laboratory conditions, indicate that very large numbers of microorganisms are usually necessary to induce disease when administered by the mouth. This is only one of the many discrepancies between laboratory and natural conditions. Many large epidemics have been traced to individual instances of pollution. In the typhoid epidemics at Butler, Plymouth, New Haven, in Nanticoke and Reading, there were collectively 3,929 cases of typhoid fever, with 361 deaths, resulting from the careless treatment of the discharges of but five individual patients.

Outbreaks due to water are usually caused by the contamination of surface supplies; less often by wells and springs. It is self-evident that the great epidemics have always been caused by polluted river or lake waters, and not by ground waters. Ground water is sometimes responsible for outbreaks of typhoid fever, especially in limestone districts, as at Lausen, Switzerland; Paris, France, etc. Usually when a well becomes badly infected it is from a nearby privy or broken sewer underground, as in the instance of the Broad Street cholera epidemic in London.

Epidemics from public water supplies result from contamination by various factors. The use of a raw water into which is continually discharged the sewage of other towns has occurred at Pittsburgh, Lawrence, and Philadelphia. A city may drink the water of a lake which has become its own cesspool, as did Chicago, Cleveland, and Burlington. The pollution may come from the wastes of individual houses, as at Plymouth, or from institutions or factories; or the pollution may come from privies situated directly over the stream or on its banks, as at Ithaca; or the pollution may come indirectly after the offending matter has been deposited on the surface of the ground, later gaining access

to the water course by the washing of rain or seepage through ground seams. In some instances epidemics originate through criminal thoughtlessness in a town that has been supplied with a pure or purified water. Thus a water pipe laid through a polluted pond may become sufficiently disjointed to permit admission of the infected water, as occurred at Baraboo, Wis., and Palmerton, Pa. The admission of polluted water to a pure city supply at any time is inexcusable. Epidemics have originated as a result of the unusual drain upon the water supply at times of fire, as in the case of Lawrence; or through failure of valves to operate, as in the case of Wilkinsburg, Pa., when the ordinary water supply was judged to be insufficient and no public warning was given of the substitution as at Newburyport; or when polluted water was furnished temporarily while the filter plant was undergoing repair, as at Lawrence, Mass., in 1902; in Brewer, in Poughkeepsie, N. Y., and Millinocket, Me. Various public wells have become infected through ground seams, and have thus caused epidemics of typhoid fever at Trenton, Newport, and Mt. Savage, Md.[1]

In addition to the usual sources of pollution of a surface water, the following, while relatively infrequent, may be particularly dangerous, for the reason that they are apt to take place near the source of supply: discharges from water-closets of railroad trains while crossing bridges or passing the banks of reservoirs and streams; picnic parties; camping parties; construction gangs; fishermen; ferryboats and other craft upon navigable streams. The large boats plying our Great Lakes may discharge dangerous and obnoxious material very near an intake.

Cholera.—Cholera in London in 1854—The Case of the Broad Street Pump.—Cholera was prevalent in London in 1854, but prevailed with epidemic intensity in the district about Broad Street. This focus was conspicuously circumscribed in area, and the disease was virulent, with great fatality. This case has become classic because it was one of the earliest instances, if not the first, in which water was proved to convey a specific disease. The circumstances were studied by Dr. John Snow and by Mr. John York, Secretary and Surveyor of the Cholera Inquiry Committee.[2] No less than 700 deaths occurred in St. James Parish during the seventeen weeks that the cholera raged. The death rate was 220 per 10,000 in the parish, which contained a population in 1851 of 36,406. In the adjoining districts the death rate varied from 9 to 33 per 10,000.

Dr. Snow made a careful epidemiological study of the outbreak and

[1] Harold B. Wood: "The Economic Value of Protecting the Water Supplies." J. A. M. A., Oct. 2, 1909, p. 1093.

[2] The complete original report is entitled "Report on the Cholera Outbreak in the Parish of St. James, Westminster, during the Autumn of 1854. Presented to the Vestry by the Cholera Inquiry Committee, July, 1855." London, J. Churchill, 1855.

compiled a statistical statement of special value, which is here given in its original form.

THE BROAD STREET (LONDON) WELL AND DEATHS FROM ASIATIC CHOLERA NEAR IT IN 1854

Date	Number of Fatal Attacks	Deaths	Date	Number of Fatal Attacks	Deaths
Aug. 19	1	1	Sept. 11	5	15
Aug. 20	1	0	Sept. 12	1	6
Aug. 21	1	2	Sept. 13	3	13
Aug. 22	0	0	Sept. 14	0	6
Aug. 23	1	0	Sept. 15	1	8
Aug. 24	1	2	Sept. 16	4	6
Aug. 25	0	0	Sept. 17	2	5
Aug. 26	1	0	Sept. 18	3	2
Aug. 27	1	1	Sept. 19	0	3
Aug. 28	1	0	Sept. 20	0	0
Aug. 29	1	1	Sept. 21	2	0
Aug. 30	8	2	Sept. 22	1	2
Aug. 31	56	3	Sept. 23	1	3
Sept. 1	143	70	Sept. 24	1	0
Sept. 2	116	127	Sept. 25	1	0
Sept. 3	54	76	Sept. 26	1	2
Sept. 4	46	71	Sept. 27	1	0
Sept. 5	36	45	Sept. 28	0	2
Sept. 6	20	37	Sept. 29	0	0
Sept. 7	28	32	Sept. 30	0	0
Sept. 8	12	30	Date unknown	45	0
Sept. 9	11	24			
Sept. 10	5	18	Total	616	616

Many of the facts of this epidemic are taken from Sedgwick's excellent account in his "Principles of Sanitary Science and the Public Health," 1902, which the student is advised to read.

It will be seen that the disease broke out with special intensity upon August 30 and declined noticeably after September 10. The pump had been removed on September 8. Dr. Snow's inquiry showed that most of the victims had preferred or had access to the water of the Broad Street well, and only in a few cases was it impossible to trace any connection with that source. Thus, with regard to 73 deaths occurring in the locality of the pump and studied especially with reference to this point, it was found that there were 61 instances in which the deceased persons used to drink the water from the pump in Broad Street, either constantly or occasionally. In 6 instances no information could be obtained, and in 6 cases it was stated that the deceased persons did not drink the pump water before their illness.

On the other hand, Dr. Snow discovered that, while a workhouse (almshouse) in Poland Street was three-fourths surrounded by houses in which cholera deaths occurred, out of 535 inmates of the workhouse

only 5 cholera deaths occurred. The workhouse, however, had a well of its own in addition to the city supply, and never sent for water to the Broad Street pump. If the cholera mortality in the workhouse had been equal to that in its immediate vicinity it should have had 50 deaths.

A brewery in Broad Street employing seventy workmen was entirely exempt, but, having a well of its own, and allowances of malt liquor having been customarily made to the employees, it appeared likely that

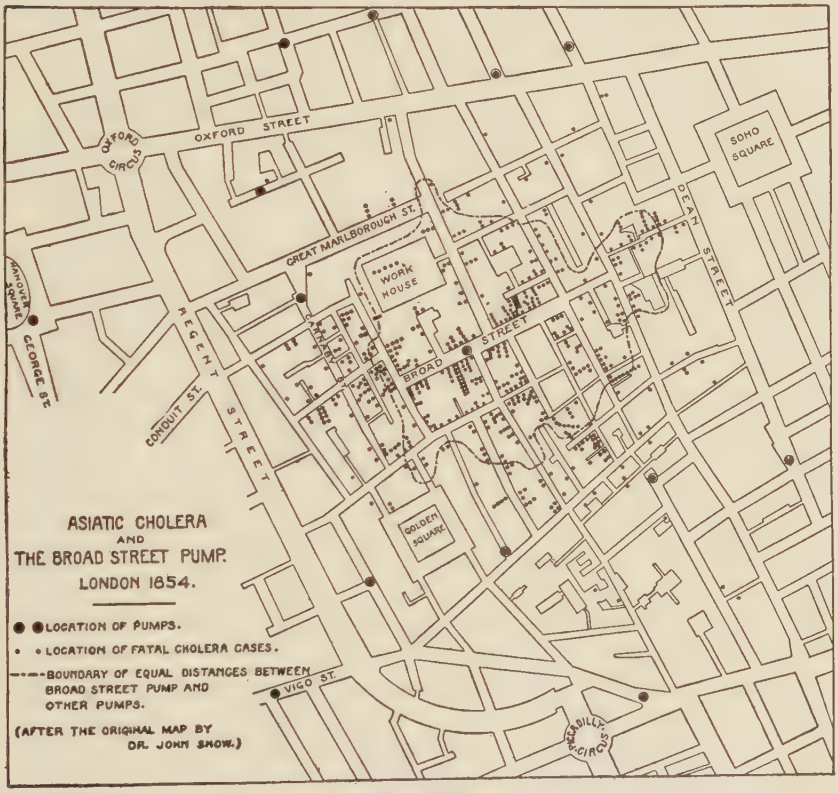


Fig. 109.

the proprietor was right in his belief that resort was never had to the Broad Street well.

It was quite otherwise in a cartridge factory at No. 38 Broad Street, where about 200 workpeople were employed, two tubs of drinking water having been kept on the premises and always filled from the Broad Street well. Among these employees eighteen died of cholera. Similar facts were elicited for other factories on the same street, all tending to show that in general those who drank the water from the Broad Street well suffered either from cholera or diarrhea, while those who did not

drink that water escaped. The whole chain of evidence was made absolutely conclusive by several remarkable and striking cases in Dr. Snow's report like the following:

"A gentleman in delicate health was sent for from Brighton to see his brother at No. 6 Poland Street who was attacked with cholera and died in twelve hours, on the first of September. The gentleman arrived after his brother's death, and did not see the body. He only stayed about twenty minutes in the house, where he took a hasty and scanty luncheon of rump steak, taking with it a small tumbler of cold brandy and water, the water being from the Broad Street pump. He went to Pentonville, and was attacked with cholera on the evening of the following day, September 2, and died the next evening."

"The deaths of Mrs. E. and her niece, who drank the water from Broad Street at the West End, Hampstead, deserve especially to be noticed. I was informed by Mrs. E.'s son that his mother had not been in the neighborhood of Broad Street for many months. A cart went from Broad Street to West End every day, and it was the custom to take out a large bottle of the water from the pump in Broad Street, as she preferred it. The water was taken out on Thursday, the 31st of August, and she drank of it in the evening and also on Friday. She was seized with cholera on the evening of the latter day, and died on Saturday. A niece who was on a visit to this lady also drank of the water. She returned to her residence, a high and healthy part of Islington, was attacked with cholera, and died also. There was no cholera at this time, either at West End or in the neighborhood where the niece died. Besides these two persons only one servant partook of the water at West End, Hampstead, and she did not suffer, or, at least, not severely. She had diarrhea."

Mr. York, Secretary and Surveyor of the Cholera Inquiry Committee, was instructed to survey the locality and examine the well, cesspool, and drains at No. 40 Broad Street. His report revealed the following condition of affairs: The well was circular in section, 28 feet 10 inches deep, 6 feet in diameter, lined with brick, and when examined (April, 1855) contained 7 feet 6 inches of water. It was arched in at the top, dome fashion, and tightly closed at a level 3 feet 6 inches below the street by a cover occupying the crest of the dome. The bottom of the main drain of the house from No. 40 Broad Street lay 9 feet 2 inches above the water level, and one of its sides was distant from the brick lining of the well only 2 feet 8 inches. This was an old-fashion drain 12 inches wide, with brick sides; the top and bottom were made with old stone. It had a small fall to the main sewer. The mortar joints of the old stone bottom were found to be perished, as was also the jointing of the brick sides, which had brought the brickwork into the condition of a sieve, and through which the house drain-

age must have percolated for a considerable period. Dr. Snow found the cesspool intended for a trap, but misconstructed, and upon and over a part of the cesspool a common privy, without water supply, for the use of the house had been erected. The brickwork of the cesspool was found to be in the same decayed condition as the drain. Dr. Snow states that, "from the charged condition of the cesspool, the defective state of its brickwork, and also that of the drain, no doubt remains upon my mind that constant percolation, and for a considerable period, had been conveying fluid matter from the drains into the well. A washed appearance of the ground and gravel flow corroborated this assumption. The ground between the cesspool and the well was black, saturated, and in a swampy condition, clearly demonstrating the fact." This evidence, while only circumstantial, is sufficient to connect the cesspool with the well, and can leave no doubt in the minds of those who study this interesting and instructive instance that the water became infected with cholera germs through this channel. It should be remembered that this outbreak occurred before the days of bacteriology, so that direct proof is not at hand. As far as could be determined, the infection of the well came from an unrecognized case of cholera in the house at No. 40 Broad Street. There were four severer cases of cholera subsequently in the same house.

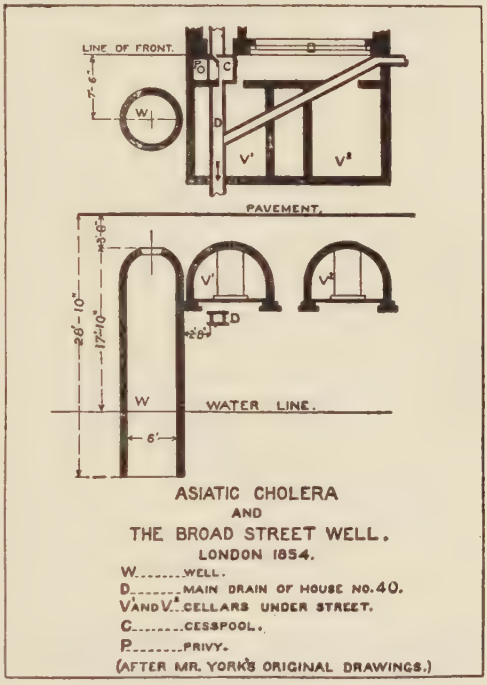


Fig. 110.

The Cholera Epidemic in Hamburg in 1892.—This epidemic stands out clearly, not only as one of the most devastating of modern times, but as one of the most instructive. The relation between the infected water and the disease was conclusively proven, and the value of slow sand filtration placed upon a strong foundation. The conditions of the epidemic were equal to those of a well-controlled laboratory experiment, and the bacteriological and epidemiological evidence corroborated each other in every essential particular.

From August 17 to October 23, 1892, a little over two months, there were nearly 17,000 cases of cholera in Hamburg (population 640,000), with 8,605 deaths. On one day during the height of the epidemic over 1,000 new cases occurred. This was a pandemic year for cholera in the sense that it showed a remarkable tendency to spread to all parts of the world. It traveled from the valley of the Ganges through Persia, to Russia, Germany, Austria, France, Belgium, Holland, and the disease was brought to our own doors and several cases occurred in New York City.

Hamburg is a separate city, and at the time of the epidemic had a population of 640,000. Altona (population 143,000) is in Prussia. Politically Hamburg and Altona are separate, but geographically and actually they form one large city. The boundary runs through a street on one side of which is Hamburg, on the other Altona. Wandsbeck (population 20,000) is a nearby suburban town. Each of these three places at the time of the epidemic had a separate water supply. Wandsbeck drank filtered water from a spring little subject to pollution. Hamburg and Altona were both furnished with water from the Elbe River, which is a grossly polluted stream. Both the cities of Hamburg and Altona rest upon the bank of the Elbe River, but Altona is below or downstream. At the time of the epidemic the intake for the water supply of each city was directly at the river front, and the sewers of the city emptied into the river at various points along the same river fronts. It will therefore be seen that Altona had Elbe River water plus Hamburg's sewage. Altona, however, first filtered this water by the slow sand process; Hamburg, however, furnished its citizens with the raw, unfiltered Elbe River water. This water was first pumped to a single reservoir, which at one time held approximately a day's supply, but had long outgrown its usefulness. It will therefore be seen that these three cities, with a homogeneous population, with the same climate, the same low-lying site, and all other conditions similar, differed only in their water supply.

During the epidemic the deaths in the several cities were as follows:

	Population	Deaths	Deaths per 10,000 Inhabitants
Hamburg	640,400	8,605	134.4
Altona	143,000	328	23.0
Wandsbeck	20,000	43	22.0

Relatively few cases occurred in Altona, and most of these were on the boundary, where the people probably had access to the raw, un-

filtered Elbe River water. In Koch's own words, "cholera in Hamburg went right up to the boundary of Altona and there stopped. In one street, which for a long way forms the boundary, there was cholera on the Hamburg side, whereas the Altona side was free from it."

Further evidence consisted in the fact that at one point close to and on the Hamburg side of the boundary line between Hamburg and Altona is a large yard known as the Hamburger Platz. It contains two rows of large and lofty dwellings containing seventy-two separate tenements and some 400 people belonging almost wholly to those

FIG. 111.—Hamburg received its water supply from the River Elbe (unfiltered) at G. The sewage of Hamburg and Altona entered the Elbe at ABC. Altona received its water supply from the Elbe at D, about 8 miles below ABC. The sand filters which purified this water were located at Blankenese. Wandsbeck had an independent water supply from a small lake.

classes who suffer most from cholera elsewhere in Hamburg. While cholera prevailed all around no single case occurred among the many residents of this court during the whole epidemic. Koch found that, owing to local difficulties, water from the Hamburg mains could not easily be obtained for the dwellings in question, and hence a supply had been obtained from one of the Altona mains in an adjacent street. This was the only part of Hamburg that received Altona water, and it was also the only spot in Hamburg in which was aggregated a population of the class in question which escaped the cholera. The source of the epidemic was traced to Russian immigrants crowded in barracks on one of the wharves pending their embarkation for the United States, and "at the time of the outbreak there were on an average about 1,000 of these people on hand all the time. Many of them came from districts in Russia which had been, and were then, suffering severely from cholera, and all were well supplied with dirty clothing and blankets,

some of which they washed while they were being detained. It is believed that among those that had arrived there must have been some mild cases of the disease, or at least some convalescents with cholera germs still in their evacuations two or three weeks after recovery. All of the sewage matters of every description from these people were discharged directly into the river at the wharf." After the Elbe River once became seeded with the cholera vibrios the people in Hamburg who drank this infected water took the disease, and their discharges, returning to the river, added fuel to the flames. A vicious circle was thus set up, so that the infection became exceedingly concentrated and intense, and as the circle was a short one the time interval was correspondingly brief and the virulence unusually severe.

The Hamburg outbreak will ever remain classic on account of the clearness of the circumstances and the fact that there is no missing link in the chain of evidence as the specific organism was readily isolated from the Elbe River water.

Typhoid Fever.—The Influence of Pure Water Upon Typhoid Fever.—The effect of an improved water supply appears to have a more favorable influence upon typhoid fever than upon any other disease. The relation between water and typhoid fever has long been known, and the attention of vital statisticians has been focused upon the improvement in morbidity and mortality of this disease following the purification of a water supply. There is now reason to believe that the good effects of a pure water in preventing other diseases may possibly outweigh the good effects in typhoid alone. The typhoid figures present such clear and often dramatic proof of the value of clean water in the conservation of health that a few of the striking tables and charts are shown upon the following pages, and should be carefully studied by the student.

For the general character of water-borne epidemics of typhoid fever see page 87. For the relation of ice and cold water to typhoid see page 837.

The table on page 823, compiled by Kober,[1] clearly shows the effect of improving the water supply in typhoid fever death rates in American cities.

From this table we learn that the combined average annual rate from typhoid fever in cities with a polluted supply was 69.4, and after the substitution of a purer water fell to 19.8 per 100,000—a reduction of 70.5 per cent. The *Bulletin* of the New York State Health Department for April, 1908, shows that the death rate from typhoid fever in ten cities of that state has been reduced 53.4 per cent. by an improved water supply. Many similar instances are cited in the literature.

[1] "Conservation of Life and Health by Improved Water Supply," George M. Kober, 1908.

TABLE SHOWING THE AVERAGE TYPHOID DEATH RATE PER 100,000 FOR A PERIOD PRIOR TO THE IMPROVEMENT IN THE WATER SUPPLY, THE AVERAGE TYPHOID DEATH RATE PER 100,000 SINCE THE CHANGE IN THE WATER SUPPLY, AND THE PERCENTAGE OF REDUCTION CAUSED BY THE IMPROVEMENT;

	Place	Average Before Improvement	Average After Improvement	Per Cent. Reduction in Death Rate
1	Albany	88.8	23.7	73.0
2	Binghamton	39.3	11.7	72.2
3	Elmira	54.9	41.5	24.4
4	Hornell	42.2	24.7	41.4
5	Hudson	64.3	31.9	50.5
6	Ithaca	67.2	14.6	78.3
7	Rensselaer	95.5	54.4	43.0
8	Schenectady	25.0	14.4	42.6
9	Troy	58.2	31.0	46.8
10	Watertown	94.7	36.9	61.8

The Typhoid Epidemic at Lausen, Switzerland.—The epidemic of typhoid fever which occurred in Lausen, Switzerland, in 1872, was the first to attract general attention, "and, because of certain peculiar conditions connected with it, and especially because of its influence upon the theory and practice of the purification of water by filtration, it deserves the most careful consideration by all students of sanitation." It is also interesting because of the remoteness and unusual method by which the infection reached the water supply. The following account of this epidemic is from the description by Sedgwick, quoting Dr. Hagler's report:

The epidemic occurred in the little village of Lausen in the canton of Basel in Switzerland in August, 1872. Lausen was a well-kept village of 90 houses and 780 inhabitants, and had never, so far as known, suffered from a typhoid epidemic. For many years it had not had even a single case of typhoid fever, and it had escaped cholera even when the surrounding country suffered from it. Suddenly, in August, 1872, an outbreak of typhoid fever occurred, affecting a large part of the entire population.

A short distance south of Lausen is a little valley, the Fürlerthal, separated from Lausen by a hill, the Stockhalden, and in this valley, on June 19, upon an isolated farm, a peasant, who had recently been away from home, fell ill with a very severe case of typhoid fever, which he had apparently contracted during his absence. In the next two months there occurred three other cases in the neighborhood—a girl, and the wife and son of the peasant.

No one in Lausen knew anything of these cases in the remote and lonely valley, when suddenly, on August 7, ten cases of typhoid fever appeared in Lausen, and by the end of 9 days 57 cases. The number rose in the first four weeks to more than one hundred, and by the end

DEATH RATES PER 100,000 OF POPULATION

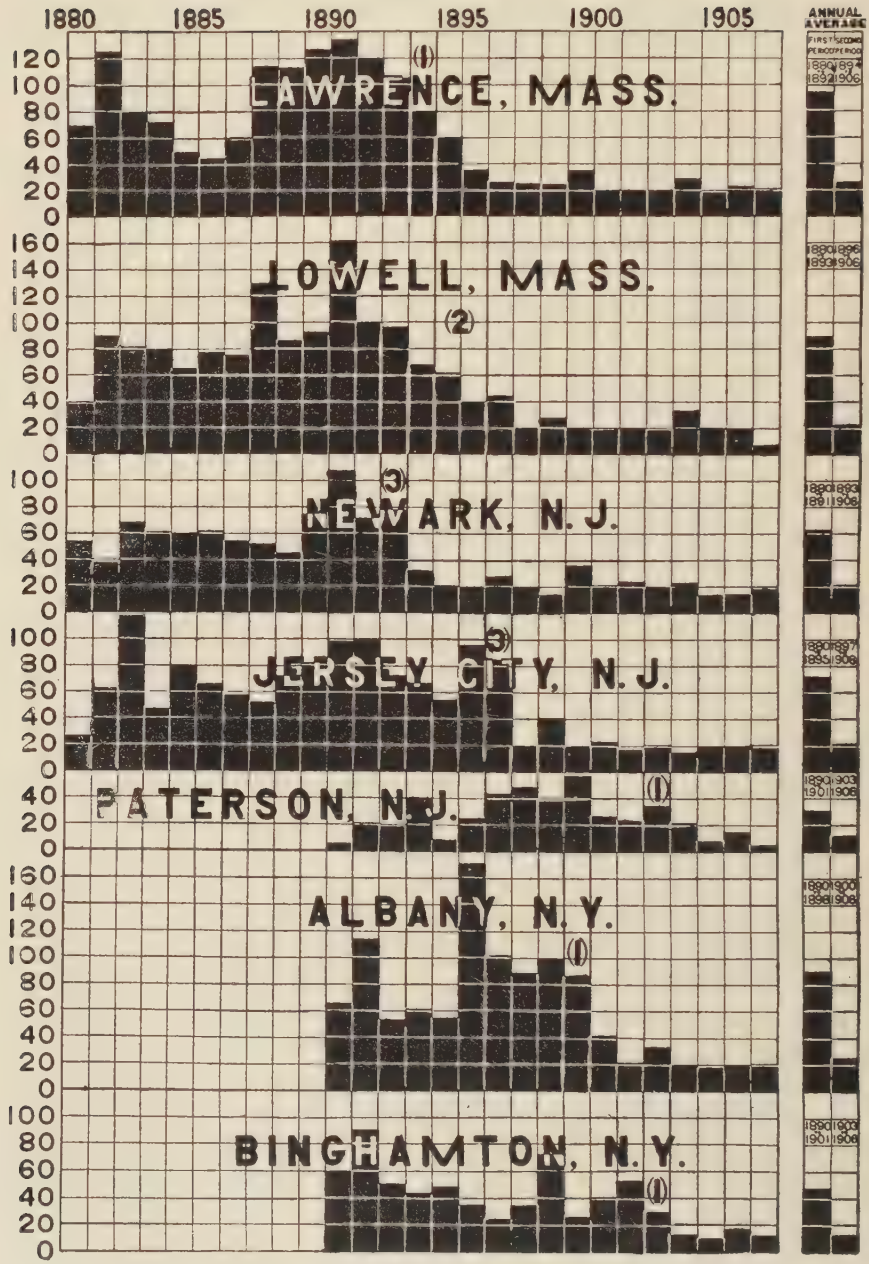


FIG. 112.—CHANGE IN WATER SUPPLY.

1.–From unfiltered river supply to filtered river supply.
2.–From unfiltered river supply to wells.
3.–From polluted river supply to conserved river supply.

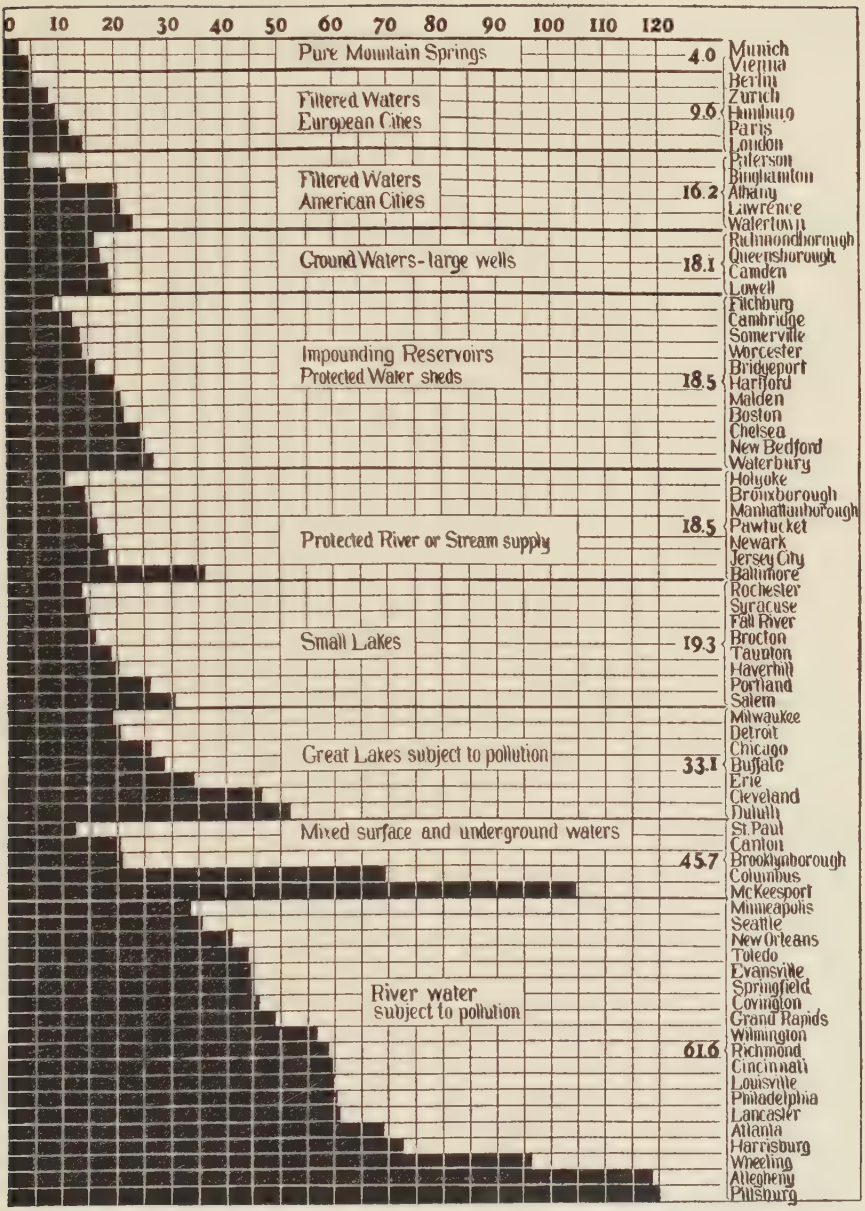


FIG. 113.—MEAN DEATH RATES FROM TYPHOID FEVER, 1902 TO 1906, IN 66 AMERICAN CITIES AND 7 FOREIGN CITIES. GROUPED ACCORDING TO THEIR DRINKING WATER. The rates for foreign cities are taken from James H. Fuertes.

of the epidemic in October to about 130, or seventeen per cent. of the population. Besides these, fourteen children who had spent their summer vacation in Lausen fell ill with the same disease in Basel. The fever was distributed quite evenly throughout the town, with the exception of certain houses which derived their water from their own wells and not from the public water supply. Attention was thus fixed upon the latter, which was obtained from a well at the foot of the Stockhalden hill on the Lausen side. The well was walled up, covered, and apparently protected, and from it the water was conducted to the village, where it was distributed by several public fountains. Only six houses used their own wells, and in these six there was not a single case of typhoid fever, while in almost all the other houses of the village, which depended upon the public water supply, cases of the disease existed. Suspicion was thus directed to the water supply as the source of the typhoid, very largely because no other source could well be imagined.

There had long been a belief that the Lausen well or spring was fed by and had a subterranean connection with a brook (the Fürler brook) in the neighboring Fürler valley; and since this brook ran near the peasant's house and was known to have been freely polluted by the excreta of the typhoid fever patients, absolute proofs of the connection between the well of Lausen and the Fürler brook could not fail to be highly suggestive and important. Fortunately, such proofs were not far to seek. Some ten years before observations had been made which had shown an intimate connection between the brook and the well. At that time, without any known reason, there had suddenly appeared near the brook in the Fürler valley below the hamlet a hole about eight feet deep and three feet in diameter, at the bottom of which a considerable quantity of clear water was flowing. As an experiment the water of the little Fürler brook was at that time turned into this hole, with the result that it had all flowed away underground and disappeared, and an hour or two later the public fountains of Lausen, which, on account of the dry weather prevailing at the time, were not running, had begun flowing abundantly. The water from them, which was at first turbid, later became clear; and they had continued to flow freely until the Fürler brook was returned to its original bed and the hole had been filled up. But every year afterward, whenever the meadows below the site of the hole were irrigated or overflowed by the waters of the brook, the Lausen fountains soon began to flow more freely. In the epidemic year (1872) the meadows had been overflowed as usual from the middle to the end of July, which was the very time when the brook had been infected by the excrements of the typhoid patients. The water supply of Lausen had increased as usual, had been turbid at the beginning, and had had a disagreeable taste. And about three weeks before the begin-

ning of the irrigation of the Fürler meadows typhoid fever had broken out, suddenly and violently, in Lausen.

In order to make matters, if possible, more certain the following experiments were made, but unfortunately not until the end of August when the water of the Lausen supply had again become clear. The hole which had appeared ten years earlier, and had afterward been filled up, was reopened, and the little brook was once more led into it; three hours later the Lausen fountains were yielding double their usual volume. A quantity of brine containing about eighteen hundred pounds of common salt was now poured into the brook as it entered the hole, whereupon there appeared very soon in the Lausen water first a small, later a considerable, and finally a very strong reaction for chlorin, while the total solids increased to an amount three times as great as before the brine was added. In another experiment five thousand pounds of flour (Mehl), finely ground, were likewise added to the brook as it disappeared in the hole; but this time there was no increase of the total solids, nor were any starch grains detected in the Lausen water.

It was naturally concluded from these experiments that while the water of the brook undoubtedly passed through to Lausen and carried with it salts in solution, it nevertheless underwent a filtration which forbade the passage of suspended matters as large as starch grains. Dr. Hägler, from whose report the foregoing facts are taken, was careful, however, to state that "it is not denied that small organized particles, such as typhoid fever germs, may nevertheless have been able to find a passage." As a matter of fact Dr. Hägler's minute account does to-day give us some indication that such germs might easily have passed from the brook to Lausen, for the turbidity of which he repeatedly speaks is evidence of the passage of particles as small as, and possibly smaller than, the germs of typhoid fever.[1]

Unfortunately this was before pure cultures of bacteria were known, and no experiments were made with suspended matters as small as bacteria. The conclusion was inevitable that although filtration had in this case sufficed to remove starch grains, it had been powerless to remove the germs of typhoid fever; and, accordingly, filtration as a safeguard against disease in drinking water fell for a time into disrepute.[2]

The Typhoid Epidemic in Plymouth, Penn.—In 1885 the mining town of Plymouth, Penn., with a population of about 8,000, suffered with a severe outbreak of typhoid fever which involved one in every eight of the inhabitants. Plymouth received its water from a mountain

[1] "Typhus und Trinkwasser," *Vierteljahresschrift für öffentliche Gesundheitspflege*, VI, 154; also Sixth Report, Rivers Pollution Commission of 1868, London, 1874.

[2] See paper by Sedgwick on "The Rise and Progress of Water-Supply Sanitation in the Nineteenth Century," *Journal New England Water Works Association*, XV, 1901, p. 330, No. 4.

brook which drained an almost uninhabited watershed. The stream was dammed at intervals and the water was stored in a series of four small impounding reservoirs. The source of the infection was traced to a citizen who spent his Christmas holidays in Philadelphia and returned home in January. He contracted typhoid; the excreta were not disinfected, but were thrown either into the frozen creek or upon its banks within 25 or 30 feet of the edge of the stream. (See map.) At this time the brook was frozen and remained so until spring. There came a thaw in March and the entire accumulation was washed into the brook and thence into the water-main. Three weeks thereafter cases of typhoid by the score made their appearance throughout the town. On some days more than 100 new cases occurred. In all 1,004 cases were reported. Some estimates placed the number at 1,500, that is, 1 in every 5 of the inhabitants. There were 114 deaths. The epidemic was limited to the houses supplied with the town water or to persons who drank of the public water supply. The distinction was particularly emphasized on one street where the houses on one side had one or more cases while the houses on the other side had none at all. The former were supplied by the town water, the latter depended upon wells.

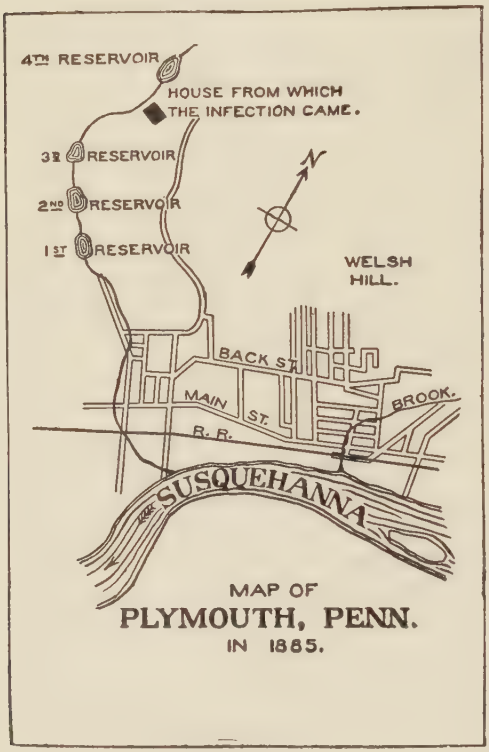


FIG. 114.

This epidemic will ever stand out in the literature as a clear-cut instance of water-borne typhoid caused by the quick transfer of virulent material from a single case. It proves further that freezing alone was not sufficient to destroy the typhoid infection, and on account of the coldness of the water it is exceedingly unlikely that any multiplication of the typhoid bacilli occurred. The infection, although greatly diluted, was nevertheless sufficiently virulent to induce the disease in most of those who drank the water. It further teaches the lesson how one per-

son is sufficient to defile the "pure waters of a mountain brook draining an almost uninhabited territory." This epidemic was the first large outbreak in America where the cause was definitely traced to the water supply. It stands out sharply in the sanitary annals of our country on account of the lessons it taught and the good influence it had in stimulating other cities to safeguard and improve their water supplies.

The Typhoid Epidemic at New Haven.—Very similar to the Plymouth outbreak was that at New Haven, Conn., during April, May, and June of 1901, when 514 cases of typhoid fever occurred, resulting in 73 deaths. The outbreak was carefully studied by Professor Herbert E. Smith, who found that it was unquestionably due to an infection of one of the sources of public water supply.

The water supply in New Haven was drawn from five distinct systems. It was all surface water and was used without filtration. One of the sources was known as the Dawson supply. Dawson Lake was a storage reservoir located on West River in Woodbridge, five miles from New Haven. Dawson Lake had an area of 60 acres and a capacity of 300,000,000 gallons. There was no direct sewage pollution upon the catchment area and the rural population was only 25 per square mile.

A mile and a half above the Dawson Lake a small stream flowed into the river, and about half a mile up this stream there was a farmhouse situated at an elevation of about 180 feet above the water in the lake. Several cases occurred in this house during January and February, 1901. The excreta was thrown into a shallow privy vault without disinfection (for the reason that typhoid fever was not at first recognized). Here they accumulated and remained more or less frozen for six weeks or more. This privy was 325 feet from the brook and 40 feet above it. On March 10 and 11 there was a heavy rainfall (2.46 inches) and a sudden thaw. The flow was so large that in spite of the intervention of the storage reservoir the water in the city was in a turbid condition on the afternoon of March 11. The typhoid fever outbreak began about 10 days later, and there seems to be little doubt that infection took place at this time. Professor Smith found that 96 per cent. of the cases that occurred were in the districts supplied with water from the Dawson Lake. (Whipple.)

This outbreak again illustrates the resistance of the typhoid infection to freezing, and the danger from a surface supply that for years may run satisfactorily. Even the storage reservoir failed in this case, as in the Plymouth case, to check the quick transfer of the infection. Had the Dawson supply been filtered or otherwise purified the epidemic could have been averted.

The Typhoid Epidemic at Ashland, Wisconsin.—This outbreak is cited from Harrington and is one of peculiar interest, in that, in addition to serving as an excellent illustration of the danger of using

the same body of water as a place for the disposal of sewage and as a source of drinking water, it was made the basis of an action at law, which established the liability of water companies and municipalities in case of sickness and death caused by the distribution and use of infected water.

The city's supply is derived from an arm of Lake Superior, Chequamegon Bay, upon which the city is situated. This bay, which is about twelve miles long, and of an average width of five, varies from eight to thirty-six feet in depth. North of the city, and extending out-

FIG. 115.

ward in a northwestwardly direction, is a breakwater constructed for the protection of the harbor against northerly gales; and between it and the city the mouth of the water intake is located about a mile from the shore. (See Fig. 115.) The sewage of the city is discharged further to the west and south. The currents in the bay follow the course indicated by the arrows in the figure, and carry the sewage toward the breakwater and over the mouth of the intake. This condition of affairs was brought to the attention of the company by the health boards of the city and state repeatedly, but without results. That the water was polluted was evident on mere ocular inspection, for it was often cloudy or markedly turbulent. During the winter of 1893-94 typhoid fever made its appearance in the city, and from the initial cases a disastrous

epidemic developed, which led to the establishment of a model filtering plant.

The action at law referred to above was brought by the widow of one of the victims. In evidence it was shown that he lived continuously in Ashland, and drank no water other than that supplied by the water company; that previous to his seizure the disease had prevailed in the city, and that the discharges from the antecedent cases had passed into the waters of the bay by way of the city sewers. The court found for the plaintiff in the sum of $5,000.

The Typhoid Epidemic in Mankato, Minn.—Mankato (population 11,553) receives its water supply from four deep artesian wells on Washington Street. Two of these wells are within from 16 to 18 feet of the pumping station. The main outlet of the sewer runs down Washington Street, emptying into the river. A great flood occurred May 20 to 24, 1908. The gate in the main trunk of the sewer was let down on the night of June 24, 1908, in order to keep the river from backing up into the sewers. This caused a backing up or stasis of the sewage, which in turn backed up into a well pit of the new artesian well near the pumping station, hence sewage was pumped into the water system. Two of the other wells and suction mains were rusty and leaked and had not been properly looked after for a number of years. Then came a sudden sharp epidemic of diarrhea, June 26. Probably 2,000 persons were affected. It soon developed that the prevailing disease was typhoid fever. The epidemic lasted from June 26 and gradually died out by Nov. 20, 1908. From July 7 to Nov. 20 464 cases of typhoid fever were reported to the Health Officer. Four hundred and one of these cases were considered primary and 57 secondary or contact cases and 6 outside or imported infection.

This water-borne outbreak of typhoid fever is particularly instructive from the fact that Delia McKeever and Kate Flanagan, administratrices of the estates of their husbands, who had died of the fever, sued the city of Mankato for damages. The city demurred to this complaint on the grounds that as a government it could not be sued and was exempt because it was carrying out a government function. The Supreme Court of Minnesota held that "the state is liable if damages can be proved." The decision of the Supreme Court in holding the city liable sets an excellent precedent which places the responsibility where it should be. Citizens are evidently as much entitled to reasonable sanitary protection as they are to police protection, or to protection from accidents at grade crossings. It is a fortunate day for preventive medicine when the principle is recognized that sanitary negligence is just as culpable as the negligence which fails to place a red flag or a red lantern to warn against a pitfall in the public highway.

The Typhoid Epidemic in Ithaca, New York.—In the winter of

1903 Ithaca, New York, the seat of Cornell University, was visited by a severe epidemic in the course of which 1,350 cases of typhoid fever occurred in a population of about 13,156. The population included about 3,000 students at the university. More than 500 homes were visited and there were 82 deaths. The epidemic covered a period of about 3 months and extended from about the 11th of January, 1903, to the 1st of April, although for several months before the epidemic began typhoid fever had been unduly prevalent. The epidemic was carefully studied by Dr. George A. Soper, who clearly showed that the disease was due to the public water supply, although the original case or cases which gave rise to the epidemic were not ascertained. Ithaca had at that time three separate sources of water supply. The larger one was derived from Six-Mile Creek and the second supply from Buttermilk Creek, and the third was an independent supply for the university. The conditions on the two streams were similar. Both streams were considerably polluted by the population which lived largely in villages bordering on the streams. The nearest of these villages was 5 miles above the intake. Soper found numerous other sources of contamination on the watershed, and some even in the city of Ithaca a few rods above the intake of the water-works where there were no less than 17 privies located on the precipitous banks of the creek. It was known that during the year previous to the epidemic there had been at least 6 cases of typhoid fever on the watershed. The typhoid epidemic in Ithaca followed a flood in the river.

One episode of the epidemic is worthy of special mention, namely, a secondary outbreak which resulted from the infection of a well. This well had become popular among the residents of a certain district at the time when the public supply came to be distrusted, and its good quality was taken for granted. But the wife of the owner was taken sick with typhoid fever during the epidemic, and her dejecta passed without disinfection through the water-closet, and into a drain-pipe which ran within three or four feet of the well. The joints of the drain-pipe were insecure; and the well water, which had probably been for some time grossly contaminated, finally became infected. As a result about fifty cases of typhoid fever and five deaths were traced to people who used this well water. (Whipple.)

The Typhoid Epidemic in Butler, Penn.—Butler, Penn. (population 16,000), had an epidemic of typhoid fever in 1903. There were 1,270 cases, that is, about 8 per cent. of the population were attacked. Infection in this case was clearly water-borne and was traced to one of various points of the stream, small tributaries, or creeks. One house in particular, provided with an overhanging privy, emptied into the creek within a short distance of the pumping station.

The Typhoid Epidemics of Lawrence and Lowell.—During the

years 1890-91 a typhoid fever epidemic occurred in Lowell and Lawrence, Mass. This epidemic illustrates with great clearness what occurs on streams which are used both as sources of water supply and as receptacles for sewage. Both cities are on the Merrimac River, which was grossly polluted by the sewage of Manchester (population 44,126), Haverhill (population 27,412), Nashua (population 19,311), Concord (population 17,004), Fitchburg (population 22,037), Newburyport (population 13,947), Marlborough (population 13,805), Clinton (population 10,424), and from other sources of pollution. In Lowell 550 cases of typhoid fever occurred from Sept., 1890, to Jan., 1891. The epidemic was carefully studied by Professor William T. Sedgwick, who made a most thorough investigation.

A short time after the epidemic in Lowell typhoid fever broke out in Lawrence, nine miles downstream, and rapidly increased. The relation between these two epidemics was most striking. Lowell discharged its sewage into the river, Lawrence drank the water without filtration. The climax of the Lawrence epidemic occurred about one month after that in Lowell. In 1892 there was a repetition of this episode. Typhoid fever in Lowell was again responsible for an increase of typhoid fever in Lawrence. As a consequence of these occurrences Lowell abandoned the river and introduced a ground water supply, while at Lawrence a filtration plant was constructed which has materially reduced the amount of typhoid fever in that city. (Whipple.)

The Typhoid Epidemics of Pittsburgh and Alleghany.—These two Pennsylvania cities are situated at the junction of the Alleghany and Monongahela Rivers, where they unite to form the Ohio. In 1900 Pittsburgh had a population of 321,616 and Alleghany 129,896. Pittsburgh takes its water from the Alleghany River at Brilliant Station, six miles above the junction of the rivers, and from the Monongahela River at a point three miles above the junction. Alleghany receives its water supply from the Alleghany River at Montrose, ten miles from the point; it is drawn from a rock-filled crib. It is practically unfiltered water. Both the Monongahela and the Alleghany Rivers are grossly polluted streams, receiving the sewage from a populous watershed; in addition the sewers of the cities of Alleghany and Pittsburgh empty directly into these streams, and on account of the rapid growth of these cities much of this sewage entered the river dangerously near to the water intakes. The records of the Board of Health show that at this time there occurred annually upward of 5,000 cases of typhoid fever.

For about ten years centering around 1900 Pittsburgh and Alleghany had the unenviable distinction of having the highest typhoid death rate of any city in this country and probably of any large city in the world. At times the rates ran above 150 per 100,000. The conditions have recently been improved by the introduction of slow sand filtration for

the city of Pittsburgh. Alleghany, now officially known as North Pittsburgh, is just being furnished (1912) with filtered water.

The Typhoid Epidemic at Chicago.—The Chicago epidemic is an illustration of a city using a lake water which is infected with its own sewage. The water in 1892 was taken from Lake Michigan opposite the city at several "cribs" which were 1.5 to 4 miles off-shore. The Chicago sewage was discharged all along the water-front, while the Chicago River penetrated the city with its north and south branches and, polluted almost beyond endurance, flowed out into the lake about midway between the upper and lower cribs. The pollution of the lake water was at times so intense that the foul river water could be traced to the intakes with the eye. This intolerable situation resulted in the building of the Chicago drainage canal, the object of which was to keep the sewage out of the lake and carry it down the Des Plaines and Illinois Rivers into the Mississippi. By the construction of this canal the flow of the Chicago River was reversed so that, instead of the sewage entering the lake and polluting the water supply, the water of Lake Michigan now flows westward to the Mississippi and to the Gulf of Mexico. During the years 1890, 1891, and 1892 typhoid fever was unusually prevalent in Chicago. In 1890 1,008 of the inhabitants died from typhoid fever, in 1891 the death toll from this preventable disease was 997 and in 1892 1,489. The present conditions in Chicago, owing to the improvements in the water supply, in the milk supply, and an attack upon the residual typhoid as contact infection, have reduced the death rate to about 12 per 100,000, which is now among the lowest death rates from typhoid fever in any large city in this country, and compares favorably with some of the European figures.

The above water-borne typhoid fever epidemics have been selected as examples. Many more may be found in the literature. Whipple, in his book on "Typhoid Fever," cites numerous instances and gives in tabular form an impressive list of such outbreaks, with references to the literature.

Dysentery.—Both bacillary dysentery and amebic dysentery may be transferred through drinking water. The infection in both types of dysentery is discharged in the feces and taken in by the mouth; there is, therefore, every opportunity for water to play the same rôle in dysentery that it plays in typhoid. However, comparatively few water-borne epidemics of bacillary dysentery have been reported; these few, nevertheless, are sufficiently conclusive to be convincing. Amebic dysentery does not occur in epidemic form, but the known facts are sufficient to incriminate water as one of the vehicles of convection.

Shiga reports outbreaks in Japan from the use of well and river water. Eldridge states that dysentery is a rural disease in Japan; the use of human feces as a fertilizer and the frequency of the infection of

the numerous small streams and wells render it preëminently a water-borne disease. The epidemic described by Duprey which occurred at Grenada Island in 1901 is one of the best examples of a water-borne epidemic of dysentery. Shiga, in Osler's "Modern Medicine," gives the following instance:

In a village called Momma-Mura, at Nobechi, in Japan, in 1900, a dysentery epidemic broke out in houses situated near each other. It was proved that the well, used by all the households suffering from the disease, was infected with the dysentery bacillus. We have also an interesting example of river-water infection in Japan. There is a village called Mitake-Mura in the district Miyagi-Ken, through which a river flows. Fishing and swimming are prohibited in it because of fish breeding. In the late summer of 1899, the prohibition having been removed, the men of the village were very glad to be allowed to fish and swim once more in the river. However, after four or five days an epidemic of dysentery broke out with 10 patients on the first day, and increasing numbers daily afterward. There were in all 413 cases, of which 115 were boys under ten years of age. After investigation it was found that there was an epidemic of dysentery in a village higher up the river, and the water had been soiled with the infected clothes.

Epidemics of bacillary dysentery in this country in institutions have not, as a rule, been associated with water.

The *Entameba histolytica,* causing amebic dysentery, was recovered by Musgrave and Clegg[1] from 17 of 61 samples of the public water supply of Manila and was found in tanks used for holding distilled water and also in many wells. Recently Allan[2] has reported a small outbreak of amebic dysentery in North Carolina due to an infected well.

Diarrhea.—Polluted waters not infrequently cause diarrhea, sometimes as widespread epidemics, sometimes as small outbreaks or sporadic cases. Whenever there is a water-borne outbreak of typhoid fever or cholera there are also a large number of cases of diarrhea and gastro-intestinal disturbances in which the precise etiological factor has not been discovered. Some of these cases may be mild instances of the major disease. Infantile diarrheas are especially prevalent at such times and very likely are due to the contaminated water. Thus Reincke states that infantile diarrhea was greatly lessened after the improvement in the water supply of Hamburg. The same phenomenon was noted by Hiram O. Mills after the filtration of the water supply of Lawrence, Mass. Sedgwick noted an excessive prevalence of both typhoid fever and diarrhea in Burlington and attributed the diarrhea to the sewage contamination of the water supply. Whipple states that in Albany

[1] Musgrave and Clegg: *Bull. 18,* Bu. Gov. Lab., P. I., 93; Rep. Bd. Health, P. I., 1904-05, 10.

[2] Allan: *J. A. M. A.,* Chicago, 1909, LIII, 1561.

there was a reduction of 57 per cent. in the mortality from diarrheal diseases after the introduction of filtration in 1898. Chapin questions whether such statistical evidence is sufficient to incriminate water as an influence to the causation of diarrheal diseases.

It is generally believed that diarrhea may be brought on by changes from a hard to a soft water; also by organic and inorganic impurities.

Numerous outbreaks of diarrhea have been attributed to the following microorganisms in water, viz.: *B. coli, B. enteritidis* of Gaertner, *B. pyocyaneus, B. proteus, B. aerogenes capsulatus* of Welch, *B. mesentericus,* and streptococci. Water containing these and other organisms is not infrequently regarded as the cause of outbreaks of gastrointestinal irritation. The symptoms vary greatly in intensity, but usually the disease is not fatal excepting in the young and feeble. The relation between the diarrhea and the water is usually based upon the fact that some species of microorganisms are found both in the water and in the stools. Corroborative evidence, such as the finding of specific agglutinins and other antibodies in the blood, lends countenance to the claim that the particular microorganism is, in fact, the cause of the complaint. While the evidence is not conclusive, it is suggestive, and in many cases doubtless correct.

Malaria.—Malaria in relation to drinking water is mentioned only for its historical interest. Laveran himself, and even Ross, considered this not improbable. Celli attempted to demonstrate this relationship by administering water, from the most malarious regions of Italy, to human beings, daily up to a month. He failed completely. According to Craig all other similar experiments have similarly proved negative, except one instance studied by Ross, in which, however, the conditions of the experiment were far from conclusive.

Laveran based his judgment upon the facts that:

(1) "There have been observed cases in which, in the same locality, persons living in identical conditions, but using drinking waters from different sources, the one group being attacked in large proportion while the other group of persons are scarcely affected at all.

(2) "In certain otherwise unhealthy localities the paludal fevers have been seen to disappear by supplying pure drinking water instead of the previously stagnant waters.

(3) "In localities otherwise healthy one can contract intermittent fever from drinking the water from an unhealthy locality.

(4) "Travelers in malarial countries have found that on boiling their drinking water they escaped the disease in a large proportion of cases."

These conclusions are especially instructive, as they illustrate some of the sources of error in epidemiological studies. Similar errors were

made in the case of yellow fever before the discovery of mosquito transmission; and in other diseases.

Yellow Fever.—Yellow fever was formerly associated with drinking water, but we now know that water plays no part in the transmission of yellow fever other than breeding the *Stegomyia calopus*. In my studies at Vera Cruz I crushed a large number of infected yellow fever mosquitoes, mixed the mass in some water which was then given by the mouth to several volunteers, with entirely negative results.

Animal Parasites.—The eggs, larvæ, or other stages in the life cycle of various intestinal parasites may enter the body in drinking water. Thus the eggs of *Ascaris lumbricoides* are discharged in the feces, which may contaminate streams and then be returned to the mouth. Some cases of infection with this parasite probably occur in this way. *Oxyuris vermicularis*, the pinworm, and *Trichiuris trichiura*, the whipworm, may similarly be contracted through drinking water. The guinea worm, *Dracunculus medinensis*, invades the skin during bathing and may, perhaps, also be contracted by the mouth in drinking water. The living embryos of this worm are liberated and find their way into fresh water. There they enter the bodies of the common fresh-water flea, *Cyclops quadricornis*, which acts as the intermediate host.

It is fairly well established that the eggs of the hookworm may be taken into the stomach through drinking water, and the same is assumed of the similar parasite of Cochin China diarrhea.

The *Bilharzia hæmatobia*, and very possibly other intestinal parasites, may likewise be transmitted through drinking water.

ICE

Ice was not suspected of being a vehicle by which infection could be spread until it was shown in bacteriological laboratories that typhoid and other cultures are not killed by freezing. Leidy in 1848 showed that water derived from melted ice contained not only living infusoria, but also rotifers and worms. MacFayden proved that the temperature of liquid air (— 315° F.) does not kill bacteria. In fact, some bacteria and molds grow and multiply at temperatures as low as 0° C.

Sedgwick and Winslow[1] (1902) were the first to make quantitative studies on the effect of freezing upon pathogenic bacteria. They used cultures of the typhoid bacillus and showed that 50 per cent. of the organisms die at the end of the first week, 90 per cent. at the end of the second week, and practically all at the end of 12 weeks. They con-

[1] Sedgwick, W. T., and Winslow, C. E. A.: (1) "Experiments on the Effect of Freezing and Other Low Temperatures upon the Viability of the Bacillus of Typhoid Fever, with Considerations Regarding Ice as a Vehicle of Infectious Disease." (2) "Statistical Studies on the Seasonal Prevalence of Typhoid Fever in Various Countries and Its Relation to Seasonal Temperature." *Mem. Am. Acad. Arts and Sci.*, Vol. XII, No. 5, Aug., 1902. Summary, *Boston Soc. Med. Sci.*, 1899-1900, Vol. IV, p. 181.

sider that we may be sure that in nature the destruction would exceed rather than fall short of these figures, for the experiments were made in a test-tube where all the bacteria are imprisoned, while in nature perhaps 90 per cent. are extruded during the purifying process of freezing.

As water crystallizes it excludes suspended matter and even dissolves substances. The extent to which water thus purifies itself depends, however, upon conditions, for under certain circumstances the impurities may be entangled or even concentrated during the process of freezing.

It is necessary to distinguish between natural ice and manufactured ice.

Natural Ice.—Natural ice should be harvested from water of good sanitary quality and handled in a cleanly manner. Even when natural ice is obtained from a polluted water the danger is greatly reduced, not only because ice purifies itself in freezing, but because natural ice is usually stored weeks and months before it is used. There are plenty of clean, fresh streams, lakes and ponds from which an abundant supply may be obtained. It is comparatively easy to protect most ponds, from which ice is harvested, from undesirable pollution. Under natural conditions the surface layer of ice contains most of the impurities and the lower layers are relatively purer, for the reason that ice grows from above downward and extrudes both suspended and dissolved matters, the surface, however, receives additional contamination from the dust, snow, flooding and other sources. It is, therefore, good practice to plane the surface of snow ice.

The fact that natural ice is usually purer than the water from which it is taken is shown by the following analyses which give the chemical and bacterial composition of natural ice and the water from which it was frozen. In this case the water was a sewage-polluted stream:

	Ice 3 to 6 Inches Thick	Water
Free ammonia	.008— .034	.46 — .084
Albuminoid ammonia	.156— .214	.146— .276
Nitrates	.05 — .20	.350— .480
Chlorin	2.0 — 3.0	4.500— 6.000
Hardness	11.0 — 28.5	57.000— 60.000
Bacteria	30. —210.	5200 — 13,000
Bacillus coli in	10 c. c.—10 c. c.	1.000— 0.100
Free ammonia	.016— .136	.006— .038
Albuminoid ammonia	.230— .726	.116— .166
Nitrates	.0 — .050	.260— .400
Chlorin	0.8 — 3.50	5.500—
Hardness	18.0 — 34.0	58.500— 62.000
Bacteria	2. — 60.	2500 — 3900
Bacillus coli in	0. — 0.	1.000— 0.1 c. c.

The chemical figures in this table are in parts per million.

The reduction in the number of bacteria is noteworthy. It will be noticed that there was no diminution, rather an increase in the free and albuminoid ammonia.

Manufactured Ice.—Manufactured ice is now universally made by the ammonia process. The condensed ammonia in expanding requires heat which it takes from surrounding objects and in this way the water is frozen. There are two distinct processes; one known as "can ice" and the other as "plate ice." In the first case the freezing takes place in rectangular cans, the water freezes from the sides of the can toward the center, and the impurities are extruded and concentrated in the core, which is often visible in a cake of can ice. In making can ice the water must first be distilled or boiled in order to drive out the air, else the resulting product will contain air bubbles. Plate ice is made by freezing water in large shallow tanks. The water freezes upon the surface and when of sufficient thickness is cut out and removed in blocks. In this method it is not necessary to distill or boil the water for the reason that the air is extruded naturally during the process of freezing. Plate ice should be made from water of good sanitary quality, especially as it is not usually stored a long time before it is used.

When ice is made from distilled or boiled water it should be above reproach. I have found, however, that manufactured ice may contain more bacteria than the water from which it was made. This is due to uncleanly methods. Thus six specimens of plate ice made from water containing 64 microorganisms per cubic centimeter and no colon bacilli gave the following results:

Number of Sample	Manufacturer	Organisms per Cubic Centimeter	Colon Bacillus
24	C. P. Co.	455	Absent
29	C. P. Co.	5,000	In 1 c.c.
26	G. Ice Co.	230	In 10 c.c.
27	G. Ice Co.	650	Absent
32	C.-S. Co.	470	Absent.
34	P. Ice Co.	8	In 1 c.c.

The laborers who work "on ice," as it is termed, scrape considerable amounts of dirt from their shoes in walking over the cans and tanks, and pollution takes place from other sources.

The chemical examination of manufactured ice may show conspicuously less total solids, less chlorin and less nitrates than found in the water from which it was made. On the other hand, it may be very high in free ammonia. This is accounted for by the fact that there is always some leakage of this gas about ice factories using the ammonia process. Sometimes ammonia occurs in such quantities as to impart a distinctly alkaline taste to the manufactured ice.

There is no excuse for uncleanly methods in handling ice that is used on or in our foods. The fact that surface impurities may be washed from a cake of ice is no reason for dragging it over sputum-laden pavements, over dirty railroad platforms, and similar methods familiar to all. The general use of ice is a modern innovation. It has come into vogue within the past 100 years. For the uses of ice as a preservative see page 474.

Properties of Ice.—Latent Heat.—If one pint of water at the temperature of 0° C. be mixed with one pint of water at 79° C. the temperature of the mixture will be the mean, 39.5° C. If, however, ice be substituted for the cold water, the whole of the ice will melt, but the temperature of the resulting mixture will not be 39.5° C., but 0° C. It therefore requires considerable heat to convert ice into water. This heat becomes latent during the process of liquefaction, and is again given off when water freezes. When the surface of water freezes the latent heat liberated by the new-formed ice raises the temperature of the remaining water, and thus retards the process of freezing or solidification. Thresh points out that were not this the case water would freeze with great rapidity in winter and the ice so formed would as rapidly melt when the weather became warmer.

In the act of freezing water expands one-eleventh of its volume, and with an almost irresistible force. Thick iron shells filled with water and securely plugged are easily burst by exposure to the cold of a Canadian winter night. Water reaches its greatest density at 4° C. It expands if heated above or cooled below this temperature. It is owing to this property that in large lakes and rivers the temperature of the deep water never falls below 4° C. during the winter. If water reached its greatest density at freezing point, which is the common property of other fluids, freezing would take place from the bottom instead of at the top. The result would be that during a severe winter our streams and lakes would become one mass of ice, which all the heat of the ensuing summer would be unable to melt.

Ice and Disease.—A search of the literature discloses but few instances of disease attributable to impurities in ice. While the experimental evidence indicates that there is a quantitative reduction of the number of bacteria in freezing, and that the imprisoned bacteria gradually die, nevertheless experience has shown that low temperatures alone cannot be depended upon to remove the danger of typhoid infection. For example, we have the water-borne epidemic in Plymouth, Pa., in 1885, presumably produced from the frozen accumulation of typhoid excrement from a single case. Very similar to the Plymouth outbreak was that at New Haven, Conn., in 1901. In only a few isolated instances, however, has ice itself been accused of being the vehicle by which the infection of typhoid fever has been spread. It appears prob-

able that milder intestinal diseases may be caused by highly polluted ice, of which the Rye Beach epidemic, carefully studied by Nichols[1] of Boston in 1875, is a point in evidence.

Park[2] (1901) described an epidemic which was believed to have had its origin in ice obtained from a pond in which it was shown that the excrement from a patient sick with typhoid fever had been thrown while the pond was covered with ice.

In the second annual report of the Board of Health of Connecticut for 1882 an interesting single case of typhoid fever is cited as probably derived from ice.

Dorange[3] (1898) described an epidemic of typhoid fever attributed to ice among eight lieutenants in a regiment stationed at Rennes in the autumn of 1895. The implication of the ice in this instance rests upon a doubtful chain of evidence, however, and no mention is made of other possible factors.

Hutchins and Wheeler[4] (1903) report an epidemic of typhoid fever in the St. Lawrence State Hospital, three miles below Ogdensburg, N. Y., which seems to have been due to impure ice. The disease was endemic in the hospital for ten years, increasing from two cases with the opening of the hospital in 1890 to forty cases in 1900. Although the water supply, tested bacteriologically and chemically, gave negative results, all observers agreed that the disease was water-borne. In December, 1900, the source of the water supply was changed to the Oswegatchie River, a small Adirondack stream supplying Ogdensburg. This practically put a stop to the disease, for there were no cases of typhoid that were not clearly contracted elsewhere until October, 1902.

Following this eight persons were attacked, seven of whom were employees in the dining-room. It seems the milk "could not have been infected." The water was excluded and other sources studied, with negative results. The ice fell under suspicion. It had recently been taken from a newly opened ice-house. The ice had been harvested from the St. Lawrence River at about the same spot as the ice previously used. It was gathered in February, and consequently had been stored for seven months. This ice disclosed a contamination of 30,400 bacteria per cubic centimeter on agar plates and 50,400 on gelatin. Of eight fermentation tubes three showed evidence of contamination by the presence of the colon bacillus.

The stock of ice was then examined. In the center of certain cakes

[1] Nichols, A. H.: "Report on an Outbreak of Intestinal Disorder Attributable to the Contamination of Drinking Water by Means of Impure Ice," *Seventh Ann. Rep.*, S. B. H., Mass., 1876, p. 467.

[2] Park, W. H.: *Virchow-Hirsch's Jahrbericht f. 1901*, p. 16.

[3] Dorange: "Epidémie de Fièvre Typhoïde dû à l'Ingestion de Glace Impure," *Rev. d'Hyg.*, Vol. XX, 1898, p. 295.

[4] Hutchins, R. H., and Wheeler, A. W.: "An Epidemic of Typhoid Fever Due to Impure Ice," *Am. Jour. Med. Sci.*, Vol. CXXVI, 1903, p. 680.

were found foreign substances in the form of black or dark brown granular matter. Examined under the microscope, this matter was found to be teeming with bacteria, from which both the colon and typhoid bacillus were isolated in pure culture.

With the discontinuance of the use of this infected ice the epidemic gradually subsided. There were in all thirty-nine cases. The evidence of this outbreak was studied by Hill, who doubted the relation of the ice to the disease.

REFERENCES

Standard Methods of Water Analysis; Report of the Committee of the American Public Health Association; second edition *American Journal of Public Health,* 289 Fourth Ave., N. Y.

Mason: "The Examination of Water." Wiley & Sons, N. Y., 1899. (Hygienic Laboratory.)

Leffman: "The Examination of Water for Sanitary and Technical Purposes." 5th Ed., Blakiston's, Phila., 1903.

Richards and Woodman: "Air, Water, and Food." Wiley & Sons, N. Y., 1901.

Whipple: "The Microscopy of Drinking Water," Wiley & Sons, N. Y.

For typical and composite analyses see also:

Clarke: "The Data of Geochemistry." *Bulletin No. 330,* Series E, Chemistry and Physics, 54, U. S. Geological Survey, 1908.

"The Municipal Water Supplies of Illinois." *Bulletin* of the Illinois State Board of Health, 1908, Vol. IV, No. 6, June.

Whipple: "Typhoid Fever." Wiley & Sons, N. Y., 1908.

Turneaure and Russell: "Public Water Supplies." Wiley & Sons, N. Y., 1907.

Hazen: "The Filtration of Public Water Supplies." 3rd Ed., Wiley & Sons, N. Y., 1900.

Thresh, J. C.: "The Examination of Waters and Water Supplies." Blakiston's, Phila., 1904.

Thresh, J. C.: "Water and Water Supplies." 3rd Ed., Blakiston's, Phila., 1901.

Mason: "Water Supply." 3rd Ed., Wiley & Sons, N. Y., 1902.

Hazen: "Clean Water and How to Get It." Wiley & Sons, N. Y.

Whipple: "The Value of Pure Water," Wiley & Sons, N. Y.

SECTION VII

SEWAGE DISPOSAL

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Importance of Speedy Removal of Fecal Matter.—The basic principle that underlies all methods of sewage disposal is to get rid of the sewage as speedily as possible, with the least nuisance to the smallest number of people, with the least damage to health or property, and at the smallest cost. Experience has shown that failure to remove human excrementitious matter from a community promptly or properly is a menace to the public health. Privies and cesspools should not be tolerated in a closely built up area. Unless more than ordinary care is exercised their existence may give opportunity for the spread of disease by insects and animals and by the pollution of local wells. Statistics show that the abandonment of privies and the substitution of sewerage systems have reduced the general death rate in many a city. Thus Dr. Boobyer has reported that at Nottingham, England, in a period covering ten years typhoid fever cases occurred in 2.7 per cent. of the houses that were provided with privies, in 0.83 per cent. of the houses where pail closets were used, and in only 0.18 per cent. of the houses that had water-closets connected with the sewers. Similarly, Dr. Porter has stated that in Stockport, England, during the years 1893-7 typhoid fever occurred in 3.4 per cent. of the houses where there were privies, but in only 1.2 per cent. of the houses that had sewer connections, these figures being based on a study of over 18,000 houses. In Munich, when sewers were constructed in 1856-9 the typhoid fever death rate fell from 242 to 166 per 100,000; later, after an improved water supply and other sanitary reforms had been brought about, the typhoid fever death rate fell to a much lower figure.

By taking special precautions against the spread of infection through the agency of flies, either by preventing their breeding or preventing them from obtaining access to fecal matter, and by closing polluted wells in crowded districts, the dangers from privies and cesspools may be greatly reduced.[1] Sometimes it is wiser to do this in villages and

[1] For the dangers of polluting the soil with feces see chapter on "Soil."

small towns than to go to the expense of introducing sewerage systems, with perhaps the attendant difficulty and expense of purifying the sewage after collection.

Ordinarily in this country sewerage systems and public water supplies are introduced in towns where the population exceeds about 3,000, and in smaller places if the population is concentrated. This is so generally true that towns that have less than 2,500 or 3,000 population are classed as "rural," the larger towns being called "urban."

Dry Earth System.—The dry earth system, much in vogue before the general introduction of the water carriage system, is now but little used; yet under some conditions it has advantages. With this method the water-closets are replaced by removable water-tight receptacles, or pails, in which the fecal matter is kept covered with dry earth, ashes, or some similar material. The pails are collected at frequent intervals, preferably daily, and a clean, empty pail substituted. The material is usually buried in the ground. For isolated houses, for temporary camps of laborers, for small scattered summer colonies, and for houses situated near streams or lakes used for public water supplies this method is satisfactory, and is often the best possible method, provided that proper care is taken by the user and the collector. Cleanliness in handling, the protection of the material against flies, regular and frequent collection, occasional disinfection of the pails, and prompt burial in proper soil are essential to success.

Water Carriage System.—So accustomed are we to present methods of sewerage that it is hard to realize that the system of water carriage of fecal matter is less than a century old. Up to 1815 the public drains of London were not permitted to receive excreta; in Boston fecal matters were rigidly excluded from the sewers until 1833; and in Paris this was the case even up to 1880.

Following the report of the Health of Towns Commission in England in 1844, water-closets were rapidly introduced, and in 1847 their connection with the sewers was required by law. The modern sewerage system, therefore, dates from about the middle of the last century. Chesbrough designed a general sewerage system for Chicago in 1855. Boston's first sewerage commission was appointed in 1875. Baltimore was without a sewerage system until within a few years, and even now, 1912, the system has not been fully completed, nor have many houses been connected with it.

The introduction of the water carriage system accomplished its purpose and effectually did away with the offensive accumulations of filth around city dwellings, but it gave rise to a series of other problems that sanitarians are now endeavoring to solve. The sewers were naturally built to discharge their contents into the nearest available body of water —into river, lake, or harbor, according to the situation of the city.

Where the streams were relatively large, no nuisance was caused by doing this, but where the streams were relatively small foul conditions soon arose, and it became necessary to reduce the amount of organic matter discharged from the sewers into them. Water supplies also became infected and in some instances great epidemics followed, while infection was spread in other minor ways. Thus the problem of the removal of fecal matter was sometimes solved at one place only to reappear elsewhere. Litigation also arose between riparian owners along the water courses, involving damages caused by the pollution of the water.

The problem has thus broadened from a local one to one in which different cities and even different states have become involved. It is to the solution of these problems of maintaining our streams and lakes and harbors in a satisfactory condition that sanitarians are now earnestly devoting themselves.

Separate and Combined Systems.—The sewers and drains of a city are used for various purposes, the two most important ones being the removal of domestic house sewage, and the rain water that falls on roofs, yards, sidewalks, and streets. Sometimes the same system of sewers is used to carry both domestic sewage and storm water. Such is called a *combined system.* Sometimes the storm water is carried in relatively large drains, or allowed to flow along in the street gutters, while the domestic sewage is carried in a *separate system* of sewers of smaller size. The choice of the two systems depends upon the local situation, but in general the following conditions control.

The combined system is the older and the one more commonly used in large cities and crowded communities, for it is cheaper than a dual system, where both separate sewers for the house sewage and drains for the storm water are required. Where the storm water can be allowed to flow off in the gutters without serious inconvenience from flooding the separate system is cheaper, as the pipes are smaller. Where the sewage must be pumped or carried long distances in pipes or purified by expensive methods the advantages lie with the separate system, as the quantity of sewage is less and its flow more constant. From the sanitary standpoint either method is satisfactory. The choice of the two systems depends upon various engineering questions involving cost, so that the matter is one that should be submitted to an engineer.

Sewerage systems consist of *house sewers* or house drains that convey the sewage to the street sewers or *lateral sewers.* These unite in what are termed *district sewers,* and the latter sometimes unite in one or more *trunk sewers* of large size. *Relief sewers* are sometimes built parallel to old sewers of inadequate capacity, and *storm sewers* are sometimes built to carry away surface water, while *underdrains* may be used in connection with the separate system to remove some of the

ground water. *Intercepting sewers* are sometimes built parallel to a stream for collecting the sewage from a number of district sewers and conveying it to a safer point of discharge. When intercepting sewers are used with the combined system they are not designed to carry all of the flow at times of storm, but are provided with overflows, so that the excess of storm water discharges into the river at various points of overflow. This is a matter of importance and one to be remembered in connection with the purification of sewage, for the quantity of sewage that passes these overflows at times of heavy rain may amount to 25 per cent. or 50 per cent. or more of the sewage, and during the course of the year may amount to from 2 per cent. to 5 per cent., or even more, of the entire sewage of the city. Such overflow water is almost never purified. At Birmingham, England, Watson has estimated that, in spite of the elaborate provisions for purification, a large part of the city's sewage is at times discharged untreated, and at Milwaukee the Sewerage Commission estimated that nearly 2 per cent. of the sewage would fail to be collected by a very liberally designed system of intercepting sewers.

Quantity of Sewage.—The volume of sewage flowing in a separate system, or in a combined system during dry weather, does not differ materially from the water consumption of the city. In small towns this may be as low as 40 or 50 gallons per capita daily, although ordinarily it is rather more than this. In large cities it may amount to from 100 to 200 gallons per capita, and more than this in extreme cases.

Intercepting sewers are commonly designed to provide for a flow of 300 to 400 gallons per capita daily. The amount of storm water depends upon climatic conditions, and for this subject engineering books should be consulted. The flow of sewage fluctuates hourly, and the maximum may be from 50 to 100 per cent. of the daily average, while greater fluctuations may be found, especially in cities where large quantities of water are used in manufacturing.

Composition of Sewage.—A city's sewage consists of the public water supply soiled with the waste products of human life and refuse from household and factory, increased by a certain amount of ground water which leaks into the sewers, and, in the combined system, by varying quantities of rain water and street wash. Disintegrating and decomposing as it flows, the sewage gradually becomes a more or less homogeneous suspension of fine particles in water, with organic and mineral matter in solution. The longer the sewage flows or stands, the more its constituents become disintegrated; fecal matter and paper become unrecognizable as such; bacteria increase enormously, and assist in the breaking down of the complex organic compounds. The oxygen originally present in the water becomes reduced and finally disappears,

so that from a fresh condition the sewage becomes first stale and then "septic." Mixed with the putrefying organic matter and the swarming hosts of bacteria harmlessly engaged in their beneficent work of destroying the organic matter, there may be also bacteria which have come from persons sick with typhoid fever, dysentery, tuberculosis, and other diseases.

Sewage is obnoxious to the senses because of its decomposing organic matter, but it is dangerous to health because of the possible presence of these pathogenic bacteria.

Among the important constituents of sewage from the standpoint of purification are urea, various proteid substances such as albumin, fibrin, casein, starch, sugar, and other carbohydrates, fats, soaps, and other organic substances. Important among the elements present in the easily decomposable matter are nitrogen and sulphur. The concentration of these substances, that is, the amount present in a given volume of sewage, depends upon the per capita volume of the sewage, and varies widely in different places. Somewhat more constant, however, are these constituents when compared with the number of persons dwelling in houses connected with the sewers.

The following figures show the approximate constituents of sewage expressed in terms of grams per capita daily and in parts per million when the volume of sewage amounts to 100 gallons per capita daily.

ESTIMATED CONSTITUENTS OF AVERAGE SEWAGE

(*After Fuller*)

		Grams per Capita Daily.[1]	Parts per Million.[2]
Oxygen consumed	Two minutes boiling	15.0	39.6
	Five minutes boiling	22.0	58.0
Nitrogen as	Free ammonia	7.0	18.5
	Albuminoid ammonia	2.5	6.6
	Organic	8.0	21.1
	Total	15.0	39.6
Chlorin		19.0	50.2
Fats		19.0	50.2
Dissolved matter	Total	136.0	359.0
	Mineral	99.0	261.0
	Organic and volatile	37.0	98.0
Suspended matters	Total	66.0	246.0
	Mineral	58.0	140.0
	Organic and volatile	40.0	106.0
Total solids	Total	229.0	605.0
	Mineral	152.0	402.0
	Organic and volatile	77.0	203.0

Bacteria, 322 billion per capita daily.

[1] These figures also indicate parts per million if the per capita volume of sewage is 264 gallons per day.

[2] Assuming a per capita volume of 100 gallons per day.

The methods of sewage analyses at present are practically the same as those used in the analyses of water. (See p. 72.) They are not in all respects satisfactory.

Ventilation and Flushing of Sewers.—The old bugaboo of sewer gas that frightened our fathers before the days of bacteriology is no longer feared by sanitarians, although its influence still pervades the antique plumbing regulations in force in many places. It is indeed desirable to keep the air of sewers from mixing with the air we breathe—the debilitating influence of all impure air should be avoided—but the danger of any one's becoming infected with the germs of disease by breathing sewer air is ordinarily so extremely small as to be quite negligible.

The water carriage system offers practically no danger to the public health during the transmission of sewage. In many cities the sewers are ventilated by allowing a free flow of air from the sewers through the house drains, the individual house fixtures only being trapped. This method is apparently safe, provided the plumbing is of substantial character. If it is not, it is better to place a trap upon the main house drain. It is believed that in the future plumbing will develop along the lines of simplicity and improved quality of materials and work, and that the present complicated system of traps and vents will be abandoned.

The catch-basins, through which the street wash enters the sewers, are trapped against the egress of sewer air. The water that stands in them is a prolific breeding place for mosquitoes. Unless catch-basins are frequently cleaned, the accumulating organic matter putrefies and the odor from it may be worse than that of the air of the sewer. Catch-basins are being omitted from some of the best designed modern sewerage systems.

Combined sewers are sufficiently flushed by the storms. Separate sewers, if laid on proper grades, need little or no flushing. It has been common in the past to employ flush tanks at the end of lateral sewers, but these are troublesome and waste much water.

STREAM POLLUTION

Sewage Disposal by Dilution.—The readiest method of sewage disposal, and the one which, until within the last few years, has been universally practiced in this country, is to allow the sewage to flow without treatment into the nearest stream or lake or harbor. This method is known as disposal by dilution. It is a proper and satisfactory method of disposal where the dilution is sufficient. It is, however, capable of abuse, and from its abuse water supplies may become polluted, oyster beds may become infected, and in severe cases streams may be so

overloaded with sewage as to become an offense to sight and smell. Properly restricted, however, the sewage is effectively disposed of, the heavy particles settle to the bottom, the organic matter is oxidized by the oxygen dissolved in the water, and the bacteria are gradually dispersed, consumed by other organisms, killed by sunlight, or otherwise destroyed. These agencies bring about the phenomenon known as the self-purification of streams.

While it is true that hygienic and sanitary considerations materially affect the use of rivers and waterways as vehicles for the reception, transmission, and ultimate disposal of sewage, the question is primarily an economic one. The power of streams to transport suspended matter and the ability of natural bodies of water to oxidize and destroy offensive substances represent a natural resource that should be utilized just as far as this can be done with safety and without offense. For each river there is a limit to the amount of permissible pollution. The reasons for this limit are not the same in all cases, but vary according to the use that is made of the water of the river, and no universal standard can be wisely set up or maintained. When the extent of the pollution is such as to affect public health in any way by any reasonable use of the river the sanitary aspect of the situation should control.

The minimum amount of water required to dilute sewage in streams is usually considered to be from 2.5 to 4 cubic feet per second for the sewage of one thousand people. The Chicago Drainage Canal was designed on the basis of 3.3 cubic feet per second for one thousand people. Rapidly flowing streams require less than this, as much oxygen is absorbed from the air. Stagnant streams may require considerably more water. The presence of certain trade wastes in the sewage may materially increase the dilution required. For example, oily matters that float on the surface and form scums may interfere with the absorption of oxygen from the air.

In lakes the relation between the sewer outfall and the intake of the water works must be carefully considered, and the dispersion of bacteria by currents induced by the wind and temperature must be studied. In harbors the effects of the tides must be taken into account.

Dissolved Oxygen in Water.—The amount of oxygen dissolved in water depends largely upon its temperature, as shown by the figures in the table on page 850.

Water near the freezing point will hold nearly twice as much oxygen as at prevailing summer temperatures. The dilution required in summer is therefore greater than in winter, and in some situations it would be logical to construct purification plants to be operated during the summer only, thus making a material saving in cost.

Sea water dissolves about 20 per cent. less oxygen than fresh water.

In order that the dissolved oxygen may be used to its best advantage,

SOLUBILITY OF DISSOLVED OXYGEN IN WATER.

Parts per Million

Temp. ° C.	Oxygen	Temp. ° C.	Oxygen	Temp. ° C.	Oxygen
0	14.70	10	11.31	20	9.19
1	14.28	11	11.05	21	9.01
2	13.88	12	10.80	22	8.84
3	13.50	13	10.57	23	8.67
4	13.14	14	10.35	24	8.51
5	12.80	15	10.14	25	8.35
6	12.47	16	9.94	26	8.19
7	12.16	17	9.75	27	8.03
8	11.86	18	9.56	28	7.88
9	11.58	19	9.37	29	7.74

it is necessary to have the sewage thoroughly and quickly diffused through the water. Otherwise the oxygen near the point of discharge may be too greatly reduced, and nuisance may result, even though there be plenty of unused oxygen near by.

Necessity of Biological Equilibrium.—It is becoming recognized that the problem of sewage disposal by dilution is largely a biological one. The decomposition and oxidation of the organic matter in sewage are brought about by bacteria, and the bacteria serve as food for protozoa and other forms of microscopic animal life. The dissolved organic matter in sewage serves as food for algæ. These algæ and protozoa are, in turn, consumed by rotifers and crustacea, while the latter form the basis of the food supply for various aquatic animals and fishes. Thus there is a continuous biological cycle. Again, animal forms require oxygen and produce carbonic acid, while plants consume carbonic acid and produce oxygen. Where these processes occur normally and with a proper equilibrium maintained between animal and plant life, offensive conditions do not result, but where abnormal conditions are produced, as, for example, by the discharge of excessive quantities of sewage or trade wastes into a stream, a depletion of the dissolved oxygen may follow, or there may be an over-production of algæ, so that the conditions become offensive. It is coming to be realized that in order to properly determine the dilution required in any particular case the conditions required to bring about this condition of biological equilibrium must be determined.

Hygienic Aspects of Stream Pollution.—Considering the hygienic aspects of stream pollution with special reference to the pollution of water supplies, it is important to remember that the typhoid fever bacilli do not multiply in the ordinary water of our streams, but, on the contrary, when discharged into water they rapidly diminish in number. After a week not more than 10 per cent. may remain alive, and after a month not more than 1 per cent.

It follows that recent pollution is the most dangerous, and that

water stored in reservoirs and lakes becomes more and more safe for use as time of storage increases. The longevity of the typhoid bacillus is much greater in cold water than in warm water. Hence, typhoid fever epidemics are more common in winter than in summer, and in northern climates than in southern climates.

PROTECTION AGAINST POLLUTION

WATER FILTRATION

Long experience in this country and a much longer experience in England and Germany have demonstrated clearly and unmistakably that polluted waters can be and are being constantly purified by means of filtration to such an extent that they are reliably wholesome. In Germany the typhoid fever death rates in the large cities have been reduced to figures far below those of American cities. In Europe it is not at all uncommon for the typhoid death rate to remain less than 10 per 100,000 for ten and even twenty years in succession, the rate not infrequently dropping as low as 3 and 4 per 100,000. There the filtration of surface water is required by law, and the efficiency of the filters is likewise required to rise to a certain fixed standard. It is worth remembering also that the streams of Germany are far from being unpolluted with sewage, and that no general attempt is made to provide sewage purification works of high bacterial efficiency. Only in case of actual epidemics is the practice of disinfection of sewage followed. The theory that water filtration is superior to sewage purification as a means of protecting water supplies against infection appears to prevail. The success of this policy has been amply demonstrated.

TREATMENT OF SEWAGE

By appropriate processes sewage can be artificially purified so that the decomposable organic matter is removed or oxidized and the bacteria removed or killed. A complete purification is not attempted even in the best conducted plants, as the processes demanded are too elaborate, too expensive, and too uncertain of results. More often the purification is incomplete, the degree of purification secured being adjusted to the particular needs of the situation. In the past sewage treatment works have been built to remove as much of the decomposable organic matter as was necessary to enable the effluent to be discharged into some waterway without causing offensive conditions. This was the case in Europe, and especially in England, where the streams are relatively small and the cities relatively large and the amounts of trade waste considerable.

In some places greater emphasis has been placed on the removal or

destruction of pathogenic bacteria, with the object of protecting oyster beds, bathing beaches, or reducing the "load" on water filters.

The degree of purification thus required varies all the way from a nearly complete purification down to a mere straining out of the grosser solids.

Fundamental Principles of Sewage Treatment.—The fundamental processes in sewage treatment are:

(1) Separation of the suspended matter from the liquid sewage.

(2) Destruction of the putrescible organic matter in the liquid sewage looking to final mineralization by the processes of oxidation and bacterial action.

(3) The transformation of the sewage sludge to a condition of stability and inertness by bacterial action, with or without oxidation.

(4) Destruction or removal of the bacteria from the liquid effluent.

The processes involved may be classified as follows:

(1) Preparatory processes, such as screens, detritus tanks, plain settling tanks, septic tanks, digestion tanks, chemical precipitation tanks, roughing filters.

(2) Purification processes, such as sub-surface irrigation, broad irrigation, intermittent filtration, contact beds, and trickling filters.

(3) Finishing processes, such as sedimentation or coarse filtration, land treatment, disinfection.

(4) Sludge disposal by digestion tanks, filter presses, drying on land, dumping at sea.

These processes are by no means clear cut. They overlap at many points; they are used singly or in all sorts of combinations.

Preparatory Processes.—Screening.—Sewage is screened to remove the larger substances that might injure pumps, clog filters, or appear as unsightly litter. Coarse screens consist of gratings of iron bars; fine screens of wire cloth. The amount of material screened from sewage varies from 0.1 to 1.0 cubic yard per million gallons of sewage, according to the fineness of the screens. It is pressed and burned under a boiler or buried in land. Screening has attained its greatest development in Germany.

Sedimentation.—Sedimentation is the most important of the preparatory processes. By allowing the sewage to flow slowly through basins in which the velocity is checked some of the suspended matter is deposited and the sewage clarified accordingly. There are five types of sedimentation basins: (1) grit chambers or detritus tanks, (2) plain settling tanks, (3) septic tanks, (4) digestion tanks, and (5) chemical precipitation tanks.

(1) *Grit Chambers.*—Grit chambers are small settling basins in which the sewage remains for a brief interval, often not more than a few minutes, and where the velocity is commonly between 10 and 30

inches per minute. They require frequent cleaning. The material collected consists largely of sand and gravel, but usually with enough organic matter to make the sludge offensive.

(2) *Plain Settling Tanks.*—Plain settling basins retain the sewage from one to twelve hours. The velocity of flow is commonly from 0.1 to 0.5 inch per minute. Sludge is removed at frequent intervals in order to prevent bacterial decomposition.

(3) *Septic Tanks.*—Septic tanks are settling tanks large enough to retain the flow of sewage from eight to twenty-four hours or longer, the velocity of flow varying from 0.1 to 0.3 inch or more per minute. The sludge is allowed to remain in the tanks for long periods, giving opportunity for intensified bacterial action to take place in the absence of oxygen; that is, under anaerobic conditions. As a result some of the solid organic matter is liquefied or gasified and the amount of sludge reduced. This process is spoken of as digestion. It is accompanied by the presence of a scum on the surface of the tank and a continual rising and falling of sludge through the liquid. The amount of solid organic matter thus digested varies from 10 per cent. to 40 per cent., being greatest in strong domestic sewage. Septic action does not materially improve the quality of the effluent. It may, in fact, make it more objectionable. Septic action cannot be depended upon to render sewage safe so far as infections are concerned.

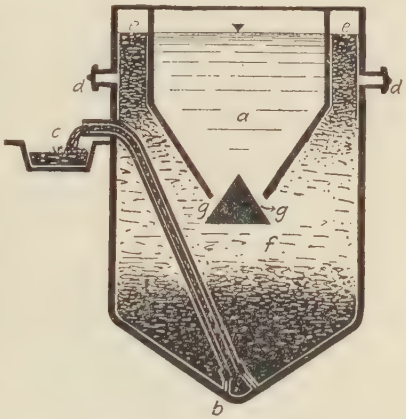

FIG. 116.—TYPICAL SECTION OF AN IMHOFF TANK.

a. Compartment for flowing sewage.
f. Sludge digestion compartment.
g. Baffle to prevent gases and sludge from rising into compartment *a*, but permitting sediment to fall into the sludge compartment.
b–c. Pipe for withdrawing sludge.

(4) *Digestion Tanks.*—The best known type of digestion tank is the Imhoff, or Emscher, tank. This is a deep septic tank divided by sloping partitions into an upper and a lower compartment, so arranged that the sewage flows through the upper compartment, while the sludge settles through openings in the partition walls into the lower compartment, where digestion takes place. The advantage of this type of septic tank is that the sludge alone is submitted to septic action without allowing the products of decomposition to mix with the flowing sewage above, while more complete digestion improves the character of the sludge from the standpoint of subsequent disposal.

The following figures show the approximate percentage of suspended matter removed by sedimentation:

PERCENTAGE REMOVAL OF SUSPENDED MATTER.

Kinds of Sedimentation	Period, Hours	Weak Sewage	Medium Sewage	Strong Sewage
Grit, or detritus tanks	1	10%	15%	25%
Plain sedimentation	6	25	40	60
Plain or septic sedimentation	12	30	50	75
Septic sedimentation	24	40	65	80
Septic sedimentation	48	50	75	85

(5) *Chemical Precipitation.*—Sedimentation may be hastened and increased by the use of chemicals. Lime, copperas (ferrous sulphate), and alum (aluminium sulphate) are commonly used. The active coagulants are the hydroxids of iron and aluminum. When the sewage itself contains the necessary amount of iron, lime only is needed.

FIG. 117.—IMHOFF TANKS AND SLUDGE DRYING BEDS, EMSCHER DISTRICT, GERMANY.

When alum is used 500 to 1,500 pounds are required per million gallons. At London the sewage is treated with 500 pounds of lime and 120 pounds of copperas per million gallons; at Worcester, Mass., with 1,000 pounds of lime and no copperas; at Providence, R. I., with 600 pounds of lime and no copperas; at Glasgow with 600 pounds of lime and 1,000 pounds of copperas.

SLUDGE DISPOSAL.—The disposal of sludge is one of the most difficult parts of sewage purification. Grit chambers collect from 0.1 to 1 cubic yard of wet sludge per million gallons of sewage; plain settling tanks from 1 to 4 cubic yards; septic tanks from 1 to 2 cubic yards. Sludge deposited in plain settling tanks contains from 90 to 95 per cent. of water; septic tank sludge, after storage, contains from 80 to 85 per cent.; chemical precipitation sludge from 90 to 92 per cent.; Imhoff tank sludge from 80 to 90 per cent. Sludge after pressing contains from 25 to 50 per cent. of water. It has some manurial value, and is

used, to some extent, on land. As a general proposition, however, the attempt to "utilize" the sludge has not met with financial success.

Purification Processes.—Sub-surface Irrigation.—For small installations a satisfactory method of disposing of sewage after sedimentation

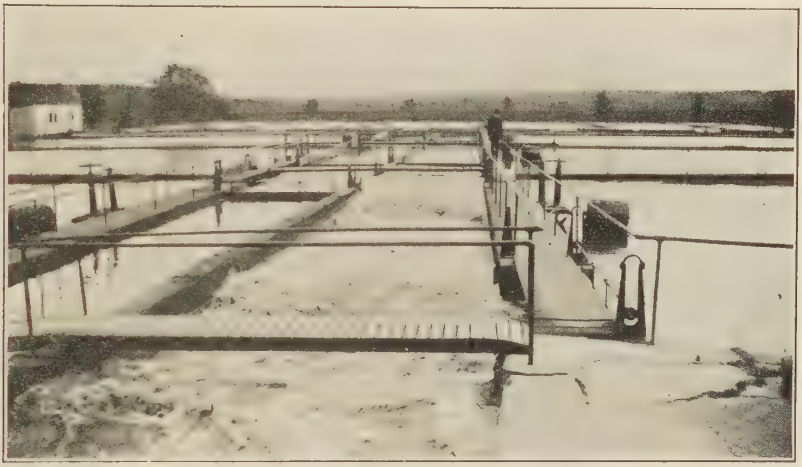

Fig. 118.—Chemical Precipitation Plant at Worcester, Mass., Inlet.

is to discharge it through 3-inch or 4-inch tile pipes laid in the ground 10 to 18 inches deep in rows 2½ to 3 feet apart. In sandy soils this method gives satisfaction, and under favorable conditions the sewage

Fig. 119.—Chemical Precipitation Plant at Worcester, Mass., Outlet.

of 150 to 250 people can be applied to an acre, the rate of application being commonly one to two gallons per lineal foot, or 20,000 to 30,000 gallons per acre daily. With tight soils larger areas are required. With clay soils the method cannot be used.

This method of sewage disposal is particularly applicable to suburban and rural conditions.

Broad Irrigation.—Broad irrigation consists in the application of crude sewage to land, making it serve as food for crops, the principal value, however, being in the water itself. It is distributed by means of ditches and other channels as in ordinary irrigation. The sewage farms of Berlin and Paris are very extensive, the Berlin farms covering nearly 20,000 acres. The rate of application varies from 3,000 to 15,000 gallons per acre daily, an acre serving for the sewage of from 100 to 300 persons. The crops raised on sewage farms frequently pay the expenses of operation, but seldom pay the interest on the investment except in arid regions, where irrigation is profitable. Broad irrigation

Fig. 120.—Triple Contact Beds at Hampton, England.

cannot be successfully used with clayey soils. The purification obtained is usually very satisfactory, both chemically and bacteriologically.

Intermittent Sand Filtration.—With this method the sewage is applied intermittently to beds of sand, especially prepared for the purpose, in such quantities that it quickly soaks away, leaving the bed exposed to the air for a period of several hours or several days, thus giving opportunity for aeration and oxidation of the organic matter. The results obtained are usually very satisfactory, provided that the filters are not overloaded. When raw sewage is applied directly to the beds the rates of application vary from 50,000 to 150,000 gallons per acre daily, the population served per acre being from 300 to 1,200. With preliminary treatment higher rates may be used, and the sewage of 1,500 to 2,000 people applied per acre. The filters are usually divided into beds by means of earth embankments which cover the distributing pipes. Often they are underdrained with tiles laid 20 to 30 feet apart

in fine material, or 100 feet apart in coarse material, their depth below the surface varying from 3 to 8 feet. Crops are sometimes grown on these beds, but agricultural operations are regarded as a secondary matter. In winter the beds are plowed into ridges or the sludge is collected into piles so that ice may form and be supported upon them, leaving channels beneath the ice by which the sewage can be distributed. After a few weeks or months the beds become clogged and it is necessary to rake the surface. At intervals the accumulated deposit on the sand has to be scraped off.

The efficiency of intermittent sand filtration is higher than that of any other process. Well operated plants are capable of removing from 95 to 98 per cent. of the suspended matter and bacteria, while the effluent

FIG. 121.—INCLINED SCREEN OPERATED BY WATER WHEEL, BIRMINGHAM, ENGLAND.

is quite clear and non-putrescible. The method is limited, however, to regions where suitable and convenient areas of sandy soil exist.

CONTACT BEDS.—Contact beds are water-tight compartments filled with porous material, such as broken stone or coke, and operated as follows: The bed is slowly filled with sewage, which has previously passed through a septic tank, and allowed to remain full for a brief period, after which it is emptied and allowed to remain empty for a longer period. A cycle commonly employed is to allow one hour for filling, two hours for contact, one hour for emptying, and four hours for rest. During the period of contact the suspended matter tends to settle upon and adhere to the exposed surfaces of the broken stone or coke, thus forming a film. While standing full septic action occurs and organic matter is absorbed by the film. During the resting period oxidation of this organic matter takes place. The purification obtained in this way is partial. Commonly, two or three contact beds are used

in series, the effluent from the first passing to the second, and that of the second to the third. The depth of contact beds varies from 2 to 6 or 8 feet, the broken stone or coke being from ½ inch to 2 inches in size. The rate of application is usually between 300,000 and 800,000 gallons per acre daily, one acre serving a population of about 5,000. When properly operated and receiving the sewage of septic tanks contact beds are capable of removing about 65 to 70 per cent. of the organic matter, 80 to 85 per cent. of bacteria, and 85 to 90 per cent. of suspended matter. Contact beds become clogged with use, and after periods varying from five to eight years it is necessary to remove the stone or coke and clean them.

Trickling Filters.—Trickling filters, otherwise called "sprinkling

Fig. 122.—Trickling Filters and Final Settling Basin and Roughing Filter at Hyde, England.

filters" or "percolating filters," consist of beds of porous material such as broken stone, coke, or clinkers upon which the sewage is sprinkled and through which it percolates to underdrains laid on a tight floor beneath. The entire bed is arranged with reference to complete aeration throughout, in order that the organic matter of the sewage may become thoroughly oxidized. The suspended matter of the sewage is not permanently retained in the beds, but is carried out in the effluent, which is turbid and requires subsequent clarification. The object of the trickling filter is to change the character of the organic matter so as to render it non-putrescible. The sewage is applied to the beds by sprinkling through fixed sprinklers or by use of traveling sprinklers, rotary or rectangular, operated by the discharging sewage or by power. The rate of application varies from 0.5 to 2.0 million gallons per acre daily, one acre of trickling filter serving a population of 10,000 or more.

The beds vary in depth from 5 to 10 feet, coarser material being used for the deeper beds. Well-operated sprinkling filters receiving the effluent from plain sedimentation or septic tanks are capable of removing from 85 to 90 per cent. of the suspended matter and from 90 to 95 per cent. of bacteria, yielding an effluent that is non-putrescible. This method is useful when sandy areas of sufficient size are not available for intermittent filtration or are too expensive.

Finishing Processes.—Disinfection of Sewage.—The best disinfectant for sewage or sewage effluents is "chlorid of lime," or bleaching powder, which is usually applied in the form of a 1 per cent. to 2 per cent. solution. The quantities required are 25 to 75 pounds per million gallons for good effluents from sprinkling filters or contact beds, 75 to

Fig. 123.—Trickling Filter at Birmingham, England.

125 pounds for poor effluents, 125 to 250 pounds for crude sewage, and 250 to 375 pounds for septic sewage, the time of contact required varying from about ½ hour to 2 or more hours. By properly applying the chemicals in these quantities it is possible to destroy from 95 to 99 per cent. of the bacteria.

Choice of Methods.—The choice of methods to be used in any case depends upon various considerations, such as the nature of the sewage to be treated, the allowable character of the effluent considered with reference to the use made of the water into which it is to be discharged, the availability of suitable areas of land at proper elevation, and finally the cost, both of construction and operation.

Where suitable areas of sandy soil are available the method of intermittent filtration is ordinarily the most satisfactory one that can be adopted. This is the case in many parts of New England and in some other parts of our country. Over much of the United States,

however, the soil is far too heavy to allow this method to be used satisfactorily, and when this is the case some of the newer methods must be resorted to, such as sedimentation followed by oxidation in trickling filters, contact beds, etc. Under some special conditions broad irrigation may be desirable, but, generally speaking, this method is falling into disuse. When the effluent is to be discharged into a stream used for a nearby supply of drinking water, or into the ocean or a harbor in the vicinity of oyster beds, disinfection may properly form a part of the process. Chemical precipitation is seldom used where the sewage is of a strictly domestic character, but it may be used to advantage when the sewage contains large amounts of trade wastes.

Methods for the purification of sewage are quite elastic inasmuch as the different processes may be combined in different ways. A study of the works that have been built in the United States during the last generation shows that not infrequently they have been made more elaborate than was necessary. Often a simpler design with a large capacity gives better results than an elaborate combination of processes of limited capacity. Important engineering problems are almost always involved in the laying out of sewage treatment works that require the services of a specialist in sanitary engineering.

Relative Bacterial Efficiency of Different Processes.—By way of recapitulation the following figures are given to show the relative sanitary efficiency of various processes employed in sewage treatment:

	Percentage Removal of Bacteria
Coarse screens	0 to 5
Fine screens	10 " 20
Grit chambers	10 " 25
Sedimentation	25 " 75
Septic sedimentation	25 " 75
Chemical precipitation	40 " 80
Contact beds	80 " 90
Trickling filters	90 " 95
Intermittent sand filters	95 " 98
Broad irrigation	97 " 99
Disinfection of raw or settled sewage	90 " 95
Disinfection of filter effluents	98 " 99

These figures are mere approximations, but they serve to show how some forms of treatment, very desirable from many points of view, have a low sanitary efficiency. The septic treatment, for example, does not greatly reduce the number of bacteria in sewage; in fact, if the period of detention of the sewage in the tank is long the numbers of bacteria in the effluent may be greater than those in the raw sewage.

Management of Sewage Treatment Works.—Proper management of sewage treatment works is as important as proper design, and is more

difficult to secure. It is a most regrettable fact that many treatment works in the United States have been badly neglected, and, in consequence, have given inefficient service. Neglect not only results in making the effluent unsatisfactory, but leaves the works themselves in an offensive condition. Neglect of small plants is more common than of plants large enough to require the entire time of one or more attendants.

Another frequent cause of failure is that treatment works are allowed to become outgrown, so that the plant becomes overloaded and the process becomes inefficient. The sewers of a city are usually designed for a long period in advance—forty or fifty years—but this is not the case with treatment works, for the reason that such works can ordinarily be enlarged when necessary. This is sound policy, for the

FIG. 124.—REMOVING SLUDGE FROM A SEPTIC TANK AT MANCHESTER, ENGLAND.

reason that the methods of purification are constantly improving, and it is desirable to take advantage of these improvements as far as possible whenever enlargement is necessary. But, if the works are to operate satisfactorily, the enlargement must be made as the tributary population increases, taking advantage of the state of the art at the time.

The purification of sewage is so largely a chemical and biological matter that it is desirable to have the works in charge of men trained in sanitary engineering, with a laboratory equipment at their disposal. Tests of the sewage before and after treatment should be made regularly in order to ascertain the efficiency of the process. Tests should also be made of the water into which the sewage is discharged. In the case of plants of large size, provided with laboratories, such tests are made daily, but in the case of plants too small to constantly employ a chemist tests should be made regularly by some controlling authority. Herein lies one of the functions of the State Board of Health.

Treatment Plants as Nuisances.—If sewage treatment works are properly designed and carefully operated, and if they are enlarged from time to time to meet the needs of the growing community, they need not be the cause of offensive conditions, but often they are, as a matter of fact, a source of nuisance in themselves. There is a natural opprobrium attached to a region where such works exist that results in a recognized deterioration of property values. The processes used for the treatment of sewage not infrequently result in odors that may be objectionable over considerable areas. Where the treatment works are entirely covered, as some kinds of works may be, little or no nuisance may result, but where, for example, the sewage is first submitted to putrefaction in a septic tank and the septic effluent then

FIG. 125.—SEPTIC TANK AND CHEMICAL PRECIPITATION TANKS AT ROCHDALE, ENGLAND.

sprayed into open air upon the surface of sprinkling filters, this exposure of the atomized liquid results in the liberation of odors that may reach distances up to perhaps half a mile from the plant, depending upon the amount and character of sewage treated, the local topography, prevailing direction of the wind, humidity in the atmosphere, and other conditions.

Frequently high winds will carry the spray itself for several hundred feet with inevitable bacterial pollution of the air. In the operation of sprinkling filters also it has been found that at certain seasons of the year swarms of flies breed in the porous beds. These are very troublesome, if not dangerous, in the immediate vicinity of such works. In considering the need of sewage treatment it is proper to balance these possible nuisances against those resulting from the discharge of unpurified sewage into a body of water. It not infrequently happens that

the installation of sewage treatment works merely substitutes one nuisance for another.

Nuisances Caused by Trade Wastes.—It not infrequently happens that the greatest nuisance in streams is due not so much to domestic sewage as to the presence of trade wastes that may be discharged into the stream directly, or that may be allowed to flow into the stream through the sewers. For example, the discharge of spent dye liquors may color the water of a stream for many miles; petroleum wastes from gas works may cause iridescent films to form upon the surface of the water, producing an unsightly appearance and increasing the odor directly, as well as indirectly, by excluding air from the water; the acid iron wastes from galvanizing works may cause a rusty discolora-

FIG. 126.—BURYING SLUDGE FROM HYDROLYTIC TANK AT HAMPTON, ENGLAND.

tion that not only imparts a brown color to the water, but paints the rocks and submerged stumps along the shores for many miles. When nuisances of this character arise it is wise and proper to install sewage clarification plants, and sometimes more elaborate works, for such nuisances cause real damage to property and to personal comfort. Trade waste pollution may interfere with the filtration of water even more than sewage itself. Illustrations of this are the paper-mill pollutions in New York State and the acid-iron wastes in Pennsylvania.

COÖPERATIVE SANITATION

What appears to be needed at the present time is some method of coöperation by which needed sanitary reforms can be brought about at least expense. It is unbusinesslike to compel the purification of the

sewage of a large upstream city in order to protect the water supply of a small city lower down, if pure water can be furnished the latter in some better and cheaper way. Legislation that clothes the State authorities with power to prevent the pollution of streams by sewage, but does not give them power to compel the purification of water or to control pollution by trade wastes, is unfortunate. It naturally leads to litigation rather than coöperation, and may retard rather than hasten necessary sanitary reforms. If our State authorities cannot be trusted in this matter it may be that a proper solution of the difficulty will be found in the establishment of district boards similar to those in England and Germany, such boards having jurisdiction over the limits of particular catchment areas. In some respects these natural hydrographic boundaries have advantages over artificial State boundaries. In the near future also our national government will doubtless take a hand in the matter. In whatever form the authority may be constituted the idea of coöperation should prevail, and ironclad rules against stream pollution should give way to a rational distribution of the burden of water purification and sewage treatment, and an equitable adjustment of cost made between the parties interested, thus decreasing the total expense of sanitary measures required and utilizing natural resources for the purification of sewage in water as far as this is safe.

If the system of water carriage of sewage continues in use the time will some day come when the sewage of all of our cities will be purified, partially or completely, and all surface water supplies filtered. It is proper to anticipate this consummation as far as our means permit, but meantime it is good business and sound common sense to spend our money first where it will go furthest and do the most good, building water filters and sewage treatment works, sometimes one, sometimes both, as they may be needed.

Adequate remedies against stream pollution from the standpoint of nuisance have been usually obtained by an appeal to the principles of common law. Cases involving bacterial pollution by sewage have been thus far too few to establish definite precedents. It will be interesting to see whether, in view of our increasing population, and especially the increasing growth of our cities, the courts will ultimately decide that the use of unfiltered river water as a source of water supply by riparian owners is a reasonable use of the water.

THE RURAL PROBLEM OF SEWAGE DISPOSAL

One of the most difficult problems of modern sanitation is to secure proper disposal of fecal matter in rural communities, at summer hotels, at temporary camps of laborers, at summer colonies at beach and mountain, and at individual houses in villages and on the farm. It is difficult

because the necessary structures are so small and simple that they have been thoughtlessly constructed, because adequate care of the processes is more or less disagreeable and therefore neglected, but chiefly because the inherent dangers have not been understood or appreciated.

FIG. 127.—CHEMICAL PRECIPITATION TANKS AT GLASGOW, SCOTLAND. LOWER END.

One of the most needed reforms, and one that is happily making progress, is that of the protected privy, that is, one where the fecal matter is received in a tight vault so constructed that the contents can-

FIG. 128.—CHEMICAL PRECIPITATION TANKS AT GLASGOW, SCOTLAND. UPPER END.

not be reached by flies, insects, rats, hens, or pigs, yet so ventilated as to prevent disagreeable odors and give opportunity for evaporation of liquids. This necessitates the liberal use of screens around the vault

and on the windows and doors, and the use of a self-closing cover for the seat. The privy vault may be constructed of concrete, with bottom and walls 3 inches to 6 inches in thickness, or the vault may be replaced with a tight, removable receptacle of metal or wood placed in a screened compartment. Properly constructed privies of this character may be located near dwellings, the only conditions being those controlling offensive odors, but this presupposes greater care than is ordinarily given to such matters. Preferably, therefore, they should be located at some reasonable distance from dwellings.

Privies that are not provided with water-tight vaults, but are so arranged that the fecal matter falls upon the soil, may be safe, so far as water pollution is concerned, if the soil is of proper character and

FIG. 129.—INTERMITTENT SAND FILTRATION BED AT BROCKTON, MASS.

if the privy is sufficiently removed from the house well; but are undesirable for other reasons. No arbitrary rules as to the necessary minimum distance of a privy from a well can be laid down, as everything depends upon the character of the soil, the slope of the ground, the elevation of the natural ground water, and the draught of water from the well. A distance of at least 25 feet should be secured with sandy soils, whenever possible, and preferably 50 feet or more. With clay soils, liable to dry and crack, and in limestone regions, liable to contain crevices in the rock, leaching privies should not be used, as wells may be polluted 100 feet or even a mile or more away.

Cesspools are holes dug in the ground to receive not only fecal matter, but also, perhaps, sink wastes and water-closet discharges. They are often lined with loose stones to prevent caving, but this permits the liquids to leach into the soil. When the soil is sandy there is no objection to this method of disposal; in fact, it is like the method of sub-

soil disposal previously described, except that the sewage is discharged into the soil below the depth where the soil bacteria are at work. This may be an important difference, however, and the oxidation of the dissolved organic matter proceeds by a slow and incomplete process. Leaching cesspools, however, should not be located near wells used for drinking water supplies. In sandy soils the danger of bacterial contamination is small if the distance is more than 25 feet, but, even so, the idea of infiltration of sewage into a well is repugnant, and often the water may be so tainted as to have a disagreeable odor, even when analysis shows it to be bacterially safe.

Ordinarily leaching cesspools should not be constructed in clay soils or in limestone regions, for they are liable to seriously pollute the

FIG. 130.—FILTER BED WITH SAND RIDGED FOR WINTER OPERATION AT BROCKTON, MASS. The ice sheet rests on the ridges. The photograph shows the accumulation of suspended matter during the winter.

ground water and are almost sure to overflow. If cesspools are necessary under such conditions they should be made water-tight and treated as septic tanks and the effluent taken care of by subsurface irrigation or some form of land treatment.

In cesspools the organic matter undergoes septic action and the amount of sludge that accumulates is often small. Nevertheless, cleaning is necessary at intervals in the case of all cesspools. The disposal of the contents is one of the most troublesome questions connected with this form of sewage disposal. The common method is to spread it upon the land as a topdressing. The work is apt to be done in the winter, when other farm work is not pressing, and not infrequently when the ground is frozen. Thus opportunity is given for fecal bacteria of human origin to be washed into a well or some public water supply. If spread

on the ground during the summer flies have access to it. If used for fertilizer for crops eaten raw, as celery or lettuce, opportunity is offered for transmission of infection by such foods. The only proper method of disposal for cesspool sludge is by burial or disinfection. In laborers' camps, and in army camps, disposal of fecal matter by cremation is practiced with advantage.

In the South, where hook-worm disease is prevalent, the scattering of human fecal matter upon the surface of the ground is one of the greatest elements of danger. The danger of transmission of infection

FIG. 131.—DISCHARGE OF SEWAGE UPON A FILTER BED AT BROCKTON, MASS.

by flies from fecal matter to food is likewise greater in the South, as the warm season is longer, so that greater care needs to be exercised in the construction and care of protected privies than in the North.

REFERENCES

1890-1910.—Annual Reports of the Massachusetts State Board of Health. (Summary of Results Obtained at the Lawrence Experiment Station during Twenty-one Years Is Given in the Report of 1908.)

1905-1910.—Contributions from the Sanitary Research Laboratory of the Massachusetts Institute of Technology. Eight volumes, containing papers by Professors Sedgewick, Winslow, Phelps, and others.

1908.—Dunbar: "Principles of Sewage Treatment." Translated by H. T. Calvert. Charles Griffin & Co., Ltd., London.

1910.—Follwell, A. Prescott: "Sewerage." John Wiley & Sons, New York.

1910.—Kinnicutt, Winslow, and Pratt: "Sewage Disposal." John Wiley & Sons, New York.

1910.—Schmeitzner, Rudolf: "Clarification of Sewage," translated by A. E. Kimberly. Engineering News Publishing Company, New York.

1911.—Kershaw, G. Bertram: "Modern Methods of Sewage Purification." Charles Griffin & Co., Ltd., London.

1912.—Elsner, Alexander: "Sewage Sludge Treatment." Translated by Kenneth Allen. McGraw-Hill Book Co., New York.

1912.—Fuller, G. W.: "Sewage Disposal." McGraw-Hill Book Co., New York.

1912.—Ogden, Henry N., and Cleveland, H. B.: "Practical Methods of Sewage Disposal."

For references to recent works for the treatment of sewage see files of *Engineering News* and *Engineering Record.*

SECTION VIII

REFUSE DISPOSAL

By George C. Whipple

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The general term "refuse" is applied to all of the solid waste material not carried by the sewers, such as ashes, rubbish, garbage, street sweepings, manure, and dead animals. The quantity of this waste material that has to be gotten rid of in a city is very large. For example, in the Borough of Manhattan, New York City, the ashes amount to about 1,200 pounds per capita per year, the rubbish 100 pounds, the street sweepings 300 pounds, and the garbage 200 pounds, the total amount of refuse being, in round numbers, a ton per capita per year. In smaller cities the per capita quantities of collected refuse are less than half of this, sometimes considerably less. The amount of garbage alone varies from less than 100 to upward of 200 pounds per capita per year. Both the quantity and character of the refuse vary with the seasons, the maximum amount of ashes occurring in the winter and the maximum amount of garbage in the summer. This fact has an important bearing on the problem of ultimate disposal.

Ashes weigh from 900 to 1,200 pounds per cubic yard, garbage from 900 to 1,100 pounds, street sweepings from 700 to 1,800 pounds, and rubbish from 150 to 250 pounds. The following figures serve to indicate approximately the constituents of the principal classes of refuse:

CONSTITUENTS OF CITY REFUSE.

	Water	Volatile Matter	Ash	Carbon	Heat Units per Pound of Refuse B. T. U.
Ashes	7–25 per cent.	8–10 per cent.	50–60 per cent.	18–25 per cent.	3,700
Garbage	70–80 per cent.	15–25 per cent.	5–15 per cent.	4–8 per cent.	2,000
Rubbish	5–15 per cent.	40–65 per cent.	5–15 per cent.	15–40 per cent.	6,000
Street sweepings	35–45 per cent.	20–30 per cent.	25–95 per cent.	18–25 per cent.	4,000

The refuse problem is to a slight extent a hygienic one, but it is more a problem of economy, convenience, and general cleanliness. Bad smells

from fermenting garbage do not directly injure the public health, yet they are an offense, and their elimination is an important matter. Ashes and street dust may irritate the eyes, nose, and throat and predispose to bacterial infection. Accumulating rubbish is not only unsightly, but may provide conditions favorable for mosquito breeding, while accumulating manure may breed flies. Garbage attracts flies and may breed them if the cans are left uncleaned from week to week, but ordinarily garbage does not stand uncollected long enough to give opportunity for the larvæ to hatch.

There are two general methods of collection and disposal of city refuse: the mixed system and the separate system. With the mixed system, which is the one most generally used in Europe, all of the refuse, ashes, garbage, and rubbish is put together by the householder in a single can, conveyed by wagon to the disposal plant, where it is all burned together and the organic matter thus destroyed. The combustible matter in the rubbish and the unburned coal in the ashes are usually sufficient to evaporate the water in the garbage, so that the material is self-consuming. This method is known as incineration, or cremation, or destruction. With the separate system the garbage, rubbish, and ashes are kept separate by the householder and collected in separate wagons and disposed of in different ways. The ashes are used for filling low land, the rubbish carried to the dump, and the garbage taken to sea and dumped or buried, or fed to hogs, or taken to a reduction plant, where it is cooked and treated for the recovery of fats and other products.

The separate system is commonly used in America, but with numerous combinations of processes of collection and disposal. Whichever method of disposal is adopted determines the manner of collection and the treatment of the refuse by the householder. The choice of the system to be used is one to be determined for each community, largely on the basis of cost. Generally speaking, an incineration plant entails a greater initial outlay than a reduction plant. Its products are ashes and steam. The ashes transported are commonly used for filling near the plant; the steam is used for power to run the works, and the excess steam is sold or converted into electricity and conveyed to places where it can be utilized to advantage. In cities where power is expensive the receipts for the sale of power may be sufficient to throw the balance in favor of this method of disposal. Where power is cheap, however, the opposite may be the case and the reduction process prove the cheaper. With the reduction process the salable products are grease and tankage. The former is sold for soap manufacture, and the latter, which consists of the solid particles of the cooked garbage, is pressed, dried, and ground, and used as a filler for fertilizers. As time goes on other useful products are likely to result from this process,

as the materials wasted or sold contain much sugar and proteid bodies.

Incineration Plants.—There are two general types of destructors. The mutual assistance type, where there are several grates and divided ash pits, the products of combustion commingling above, thus combining several furnaces into one, and the separate unit type.

The temperature of combustion varies from about 1,200° to 2,000° F. and the capacity is from 1,200 to 1,500 pounds of mixed refuse per day for each square foot of grate surface. Each pound of mixed refuse is capable of evaporating from one to two pounds of water. So-called cremation plants are operated at lower temperatures and are less satisfactory.

The best illustration of an incinerator in this country is the one recently constructed at Milwaukee. This was designed by Dr. Rudolph Hering, and a description of it may be found in the *Engineering News* for August 26, 1910. It has a capacity of 300 tons of mixed refuse per day. The Milwaukee incinerator receives street sweepings and manure as well as ashes, rubbish, and garbage. The manure has been found fully as difficult to burn as the garbage, and on general principles it would appear to be wasteful to dispose of it in that way. With a well-arranged incinerator there are practically no objectionable odors and very little disagreeable smoke.

Reduction Plants.—This method of garbage disposal is used in many of our largest American cities, including New York, Boston, Buffalo, the plants as a rule being owned and operated by private companies under contract with the city. Recently an excellent plant of the reduction type has been constructed by the city of Columbus and is now operated by the city. A description of this plant may be found in the *Engineering Record* of November 19, 1910. When the garbage reaches a plant of this type it is sorted to remove foreign substances, such as tin cans, glass bottles, etc., and conveyed to a series of digestors, where it is cooked for from six to ten hours under pressure of about 60 pounds. It then passes through presses which separate the water and fats from the solid part, called tankage. The water and grease are allowed to pass through settling tanks, where the grease is skimmed off the top. The water flows away to the sewer or is evaporated, and the solids added to the tankage. The latter is sometimes treated for fat recovery by the use of hot naphtha. Ultimately the tankage is ground and dried and used as a filler for fertilizers. The per cent. of grease recovered may amount to from 1 to 3 per cent. and the marketable tankage to about 20 per cent. of the garbage. Unless a plant of this type is well designed and carefully managed offensive odors will result, but these can be almost completely done away with if proper precautions are taken.

Feeding Garbage to Hogs.—In many small cities, especially those of New England, the garbage is fed to hogs. This requires frequent collection and careful management at the piggery. If the garbage is sterilized with steam, and if the feed is supplemented with grain, and the garbage feed stopped a few weeks before the hogs are killed, there seems to be no sanitary objection to this method, while it may be a profitable one on account of the large food value of the garbage.

Collection of Garbage.—From a sanitary standpoint, and even from the standpoint of nuisance, the problem of garbage collection is even a more difficult one than that of garbage disposal. A strong argument in favor of the incinerator method is that the method of mixed collection can be carried on with less nuisance than separate collection. When garbage is mixed with the ashes in a single can the water of the garbage is absorbed by the ashes, fewer flies are attracted to it, and the odor is reduced. The absorption of water by the ashes also tends to reduce the dust nuisance. Mixed collection is also more economical, as fewer carts are required and collections need not be as frequently made. Much depends, however, upon local conditions.

Garbage disposal plants are best located near the outskirts of the city, where no nuisance will result. This ordinarily involves a long haul. If favorable opportunities exist for dumping ashes within the limits of a short haul the separate collection of refuse may prove the cheaper. At Minneapolis the householders are required to wrap each day's garbage in paper. This method is said to be very satisfactory.

REFERENCES

1905.—Hering, Rudolph: "Review of General Practice of Disposal of Municipal Refuse." Transactions American Society of Civil Engineers, Vol. LIV, Part E, p. 263.

1906.—Parsons, H. deB.: "Disposal of Municipal Refuse." John Wiley & Sons, New York.

1906.—Venable, W. M.: "Garbage Crematories of the United States." John Wiley & Sons, New York.

1910.—Morse, W. F.: "Collection and Disposal of Wastes." Published by the *Municipal Journal,* New York.

1911.—Greeley, Samuel A.: "Investigations for Municipal Refuse Disposal." Proceedings New Jersey Sanitary Association, November, 1911.

SECTION IX

VITAL STATISTICS

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In the broadest sense vital statistics may be defined as the statistics relating to any form of life or to the growth and development of any living organism. In this sense they may constitute a part of biometrical methods as applied to biological problems of every character, dealing both with plant and animal life. In the more limited and usual sense, however, vital statistics are the statistics based upon the chief events of human life, and the events that are commonly studied by statistical methods are births, marriages, and deaths. Of minor importance are stillbirths, now usually distinguished from living births, and also from deaths of persons born alive.

The statistical method involves the study of masses or considerable numbers of events rather than that of individual or few cases. Nothing is more uncertain than the prediction of the future life of an individual at birth, but the probability that a certain proportion of individuals will die in infancy or reach adult years, will be married or remain single, will succumb to certain diseases or will survive to approximately a certain age, may be established within reasonable limits by the observation of large numbers of similar cases existing under like conditions. Changes occur in the vital statistics of large groups, both suddenly and of a periodic character, but on the whole there is a considerable stability among the averages shown for cities, states, and countries, and the trend of future events may often be anticipated from the results of the past.

Unfortunately, perhaps, statistics are obliged to deal to a considerable extent with figures and tables. This fact may account for a certain degree of unpopularity of the subject because arithmetical operations are distasteful to many minds. There is much drudgery connected with the work of statistical compilation, and it is, of course, very necessary that each step connected with the collection and com-

pilation of data be performed with precision and accuracy, as otherwise the results and conclusions based thereupon may be vitiated. It follows, therefore, that vital statistics are not infrequently neglected in the work of a public health service, and perhaps relegated to the charge of persons unfitted by training or taste properly to discharge the important duties of registration officials.

Necessity of Vital Statistics in Public Health Work.—The first duty of a newly appointed officer of public health should be to inform himself thoroughly as to the character and conduct of the vital statistics of the state or city of which he has charge. The movement of the vital statistics of the past should be examined, and he should remember that the ultimate judgment of the success of his work and of the methods of sanitary betterment that he may introduce will be based upon the careful analysis of the vital statistics collected during his term of office. Dr. Hurty has well called vital statistics the "bookkeeping of humanity," and the health officer, charged with control over the precious treasure of human life, must again and again appeal to the vital records to show how well he has discharged his trust.

There is a danger here. The health officer must be entirely candid in his use of vital statistics, must thoroughly understand the fallacies to which he is liable by the improper use of registration data or by the use of imperfect or inaccurate returns. No great degree of mathematical attainment is necessary for some of the most important practical applications of vital statistics. The ordinary ratios or "rates" employed in vital statistics are as easily computed and understood as the "percentages" so familiar to the baseball public. A little common sense in avoiding comparisons of data that are not comparable, and absolute honesty in accepting and using the results—if based upon efficient registration—are alone necessary to make the vital statistics of indispensable service in public health work.

It should not be necessary to warn against the tendency to warp the statistical data in the direction of preconceived or desired conclusions. The compilation of vital statistics can be manipulated in many ways, and by overestimates of population or exclusion of deaths for various reasons the death rates may be, apparently, reduced so that boastful claims of the "healthiest city" may seem to be justified. Such claims are usually open to question, and frequently will be found to depend either upon grave deficiencies in registration, the unjustifiable omission of certain deaths, overestimates of population, or, perhaps most frequently of all, the utter ignoring or lack of knowledge of the fact that general or crude death rates are unreliable criteria of sanitary efficiency, and that the age, sex, or other peculiar constitution of the population must be taken into consideration. The most crying needs of American registration at the present time are: (1) better registration,

that is, more complete registration, especially of births, and (2) practical knowledge of the uncertainty of the crude or general death rates as compared with death rates properly corrected for sex, age, or other modifying constitution of the population.

Neglected Condition of Vital Statistics in the United States.—The United States is exceptional among the countries of the civilized world in that there are no national system of vital statistics and no complete records of births and deaths for the country as a whole. This results from the fact that the control of registration is entirely a function of state government, or of municipal government in states where no general laws on this subject are in force.

Reports on vital statistics have been published by the Federal government for many years. The subject was first introduced in the Seventh Census and a report on the mortality statistics of the United States for the census year ended May 31, 1850, was published in 1855. This report was based on the deaths collected by the assistant marshals while enumerating the population. It will readily be understood that it is quite impossible to obtain a complete list of deaths by this method, and the statistics were far too imperfect to afford any reliable rates. Nevertheless, the plan of attempting to collect deaths by enumeration after the close of the year to which they relate was continued for successive censuses until it was finally done away with in the law for the Thirteenth Census (1910).

Year	Population of Continental United States	Population of Registration Area		Deaths in Registration Area	
		Number	Per Cent.	Number	Rate per 1,000 Population
Census year 1879–1880	50,155,783	8,538,366	17.0	178,645	19.8
Census year 1889–1890	62,622,250	19,659,440	31.4	386,212	19.6
Census year 1899–1900	75,994,575	28,807,269	37.9	512,669	17.8
Calendar year 1900		30,765,618	40.5	539,939	17.6
Calendar year 1901	77,747,402	31,370,952	40.3	518,207	16.5
Calendar year 1902	79,365,396	32,029,815	40.4	508,640	15.9
Calendar year 1903	80,983,390	32,701,083	40.4	524,415	16.0
Calendar year 1904	82,601,384	33,345,163	40.4	551,354	16.5
Calendar year 1905	84,219,378	34,052,201	40.4	545,533	16.0
Calendar year 1906	85,837,372	41,983,419	48.9	658,105	15.7
Calendar year 1907	87,455,366	43,016,990	49.2	687,034	16.0
Calendar year 1908	89,073,360	46,789,913	52.5	691,574	14.8
Calendar year 1909	90,691,354	50,870,518	56.1	732,538	14.4
Calendar year 1910	92,309,348	53,843,896	58.3	805,412	15.0

Vital statistics should be collected only by immediate *registration,* as distinguished from enumeration after the close of the period covered. Many states established systems of registration about the middle of the last century, but of these only two, Massachusetts and New Jersey, were found to possess returns of sufficient value for inclusion in the registration area for deaths which was established in the mortality reports of

the Tenth Census (1880). This area, which was based upon the requirement of approximately complete registration enforced by burial permits, embraced only these states and certain cities in other states with an aggregate population of 8,538,366 persons, or about one-sixth (17 per cent.) of the estimated population of the continental United States. The growth of this area, together with the deaths and death rates for succeeding years, is shown on page 876.

For the year 1910 the registration area for deaths had thus increased to a population of 53,843,896, or nearly three-fifths (58.3 per cent.) of the total estimated population of the country in the middle of 1910. The following list comprises the registration states for that year:[1]

California	New Jersey
Colorado	New York
Connecticut	North Carolina (municipalities
Indiana	of 1,000 population and over)
Maine	Ohio
Maryland	Pennsylvania
Massachusetts	Rhode Island
Michigan	Utah
Minnesota	Vermont
Montana	Washington
New Hampshire	Wisconsin

Returns were also received from the District of Columbia (city of Washington) and forty-three other cities in non-registration states. The registration of deaths is not equally efficient in all of these states. Some of them are sparsely settled and the returns for certain counties are practically worthless. This fact is indicated in the annual report on mortality statistics prepared by the Bureau of the Census, and it is a question whether it would not be preferable to omit entirely the reports for counties in which the State authorities appear to be unable to enforce the law. The total population belonging to such areas is small, however, and the general completeness of the returns for certain states may not be materially depressed thereby.

Registration of births is more difficult than registration of deaths. This is proved by the fact that after many years of experience with the operation of registration laws no state or city in the United States claims complete birth registration. It is not difficult to obtain by enumeration or imperfect registration a considerable proportion of the births that occur in a given area. Up to the limit of one-half or perhaps two-thirds of the actual births the collection of births may be as easy as or even easier than that of deaths. But such returns are worthless for statistical purposes, however useful the records may be for

[1] Kentucky and Missouri were added for 1911.

legal purposes. When complete returns of deaths are obtained by means of that essential requirement the burial permit it invariably happens that the completeness of the registration of births does not rise to the level attained by the registration of deaths. Consequently, it was not possible to establish a registration area for births as early as that for deaths (1880), and the tentative birth registration area established for 1908 includes a much smaller reporting population than the death registration area. The following table shows the births and birth rates for this area, which represents about one-fourth of the aggregate population of continental United States:

Year	Estimated Midyear Population	Births (exclusive of stillbirths)	Birth Rate per 1,000 Population
1908	21,296,119	547,665	25.7
1909	21,759,262	543,185	25.0
1910	22,222,404	555,486	25.0

The figures for 1910 are provisional, but approximately complete; that is, so far as the registration returns are accurate for the areas included. These are the six New England states, Pennsylvania, Michigan, the District of Columbia, and New York City.

Collection of Vital Statistics.—The vital statistics of greatest importance to the sanitary administration, namely, those of births and deaths, must be collected under a *registration* law. The births and deaths must be registered immediately or very soon after their occurrence, or some will be omitted and the records and statistics will be inaccurate. For a long time the movement for better legislation with respect to vital statistics in the United States proceeded without any general direction and supervision, and as a result of the lack of a definite policy and understanding of the necessary provisions of an adequate registration law many bills were presented to State legislatures, and some of them were given effect as laws, that were predestined to failure in practical operation.[1] Some of these laws, as, for example, the recent Tennessee enactment, may prove to be of service in preparing the way for better laws, but others stand in the path of progress and remain obstacles to effective legislation.

About 1902 the question of the improvement of registration legislation was taken up in a practical way by a special committee of the American Public Health Association and, in conjunction with the Bureau of the Census, a circular was issued in which certain essential principles were defined as necessary, according to American experience, for successful results in the registration of deaths. The committee and

[1] An improved law was recently enacted in Tennessee, which will soon be effective.

the Bureau of the Census did not go so far as to define the requirements for successful birth registration because there was, in fact, no complete birth registration in the United States upon which to base their recommendations—nor to draft bills embodying those requirements. It seemed to be a distinct step in advance to point out some solid basis, however limited, which would serve as a foundation upon which to build better laws.

Later a bill was drafted for the registration of deaths, and subsequently, in order to carry out the desire expressed by Congress for cooperation by the state authorities in the more effective registration of births and deaths, drafts of a bill for the registration of births and for the registration of both births and deaths were prepared.

The essential principles upon which these drafts were based, having stood the test of practical criticism in the construction and administration of registration laws, were adopted as "Rules of Statistical Practice" by the American Public Health Association. The requirements for effective registration of births and deaths may be compared by means of the following tabular statement:

NECESSARY PROVISIONS FOR THE REGISTRATION OF DEATHS AND BIRTHS

DEATHS	BIRTHS
1. Deaths must be registered immediately after their occurrence.	1. Births must be registered immediately after their occurrence.
2. Certificates of death should be required.	2. Certificates of birth should be required.
3. BURIAL OR REMOVAL PERMITS are essential to the enforcement of the law.	3. SOME CHECK is necessary to secure enforcement of the law.
4. Efficient local registrars are necessary.	4. Efficient local registrars are necessary.
5. The responsibility for reporting deaths to the local registrars should be fixed.	5. The responsibility for reporting births to the local registrars should be fixed.
6. The central registration office should have full control of the local machinery, and its rules should have the effect of law.	6. The central registration office should have full control of the local machinery, and its rules should have the effect of law.
7. The transmission and preservation of returns should be provided for.	7. The transmission and preservation of returns should be provided for.
8. Penalties should be provided *and enforced.*	8. Penalties should be provided *and enforced.*

THE MODEL REGISTRATION LAW.—Much of the improvement in methods of registering vital statistics in the United States has been

due to the active interest and coöperation of the medical profession. The American Medical Association has taken an especially prominent part in this movement and has circulated literature upon the subject and copies of what is known as the "model bill" or "model law" drafted in accordance with the essential requirements of registration. The following is an extract from the *American Medical Association Bulletin,* September 15, 1910:

"In *The Bulletin* for January 15, 1909, appeared the model bill drafted by a committee appointed at the 1906 conference on medical legislation. At the 1907 conference held in Chicago in December of that year the committee submitted its report, which consisted of a model bill patterned, in the main, after the Pennsylvania law. This bill has since been endorsed by the Census Department of the United States Government, the Section on Hygiene and Sanitary Science, and the House of Delegates of the American Medical Association, the American Public Health Association, the American Statistical Association, the Committee on Uniform Laws of the American Bar Association, the American Association for the Study and Prevention of Infant Mortality, and the general officers of the American Federation of Labor, as well as by numerous local and state associations and other public health bodies. It can therefore be said to represent the combined judgment of all those interested in securing better vital statistics legislation and registration for the United States, as well as the knowledge and experience of those best qualified to speak with authority on the subject. As this bill has been carefully prepared, it is especially urged that no changes be made in its verbiage or provisions, as apparently slight and unimportant changes may seriously affect the operation of the law."

The best general idea of the mode of operation of a modern registration law may be obtained by a careful examination of the provisions of this bill [1] or of the state laws, e. g., those of Pennsylvania, Ohio, Missouri, Kentucky, that are practically identical with it.

The administration of a registration law is a matter of infinite detail. The State Registrar must be in constant touch with his corps of local registrars scattered throughout the state. It is extremely important in the organization of the system that there shall be a sufficient number of such local registrars and so distributed in each county that there will be no great difficulty in the carrying out of the essential requirement of the law, namely, that all deaths shall be registered and

[1] Copies of this bill can be obtained from Dr. Frederick R. Green, Secretary, Council on Health and Public Instruction of the American Medical Association, 535 Dearborn Avenue, Chicago. The bill has been again revised by a special committee composed of representatives of the American Medical Association, American Public Health Association, American Bar Association, and Bureau of the Census, with aid from the committee on vital and penal statistics of the conference of Commissioners on Uniform State Laws and the Children's Bureau.

permits for burial or removal issued in advance of any disposition of the body. If this requirement is neglected in a few cases the number of violations will increase and soon the law will become a dead letter. But in sparsely settled districts the difficulty of communication may be very great at times, hence some provision is necessary as a safety-valve, especially during the first trial of such a law in a state in which no formalities have been observed relative to the burial of the dead. A plan whereby any registrar may issue a provisional permit for deaths not occurring in his district, afterwards seeing that the certificate is filed with the proper local registrar in time for his monthly return, or a special exemption for sparsely settled districts, with requirement of return within ten days after death, may be employed. A practical difficulty is found in rural districts where no undertakers are employed, interments being made by the relatives or friends of decedents. To secure complete returns under such circumstances will require long and patient education of the people as to the importance of such records, and also some check upon the sale of coffins by furniture dealers, so that a blank certificate and a copy of the law requiring registration may accompany each one sold.

The enforcement of the law for the registration of births is especially difficult because there is no ready check, analogous to the burial permit for deaths, which will indicate at once that the law has not been complied with. In some cities special enumerations of births are made and the registration records are supplemented by the additional births thus found. Local registrars may also "keep tab" on births reported in the press or otherwise coming to their knowledge, but probably the most effective method is the systematic examination of all returns of deaths of infants under one year of age in order to see whether their births were duly registered. For example, it is stated in the weekly vital statistics report for the District of Columbia, October 14, 1911, that "there were 14 District-born babies who died during the week covered by this report. Of these 11 had their births reported. The other three are now being investigated with a view to ascertain, if possible, who is responsible for the failure to make these reports." In New York City, beginning in October, 1910, many physicians and midwives were prosecuted and fined for failing to register births. A state inspector is constantly employed in Pennsylvania and has been instrumental in securing many thousands of births that would otherwise not have been recorded. Success depends mainly upon the *enforcement* of the law by means of the penalties provided therein, and if the law is not enforced it does little good to ascertain the fact of violation and receive the more or less hackneyed excuses of the delinquents. So much injury is done to the legal and personal interests of children and their parents by the unjustifiable neglect of physicians

and midwives who are charged with the duty of reporting births that it would seem that public sentiment should demand rigorous enforcement of registration laws.

The compensation of physicians and midwives for the reporting of births is not provided for in the model law recommended by the American Medical Association. This may perhaps be taken as an expression of medical opinion in general throughout the country, but in some localities the sentiment of the profession is urgent for a small fee, to be paid out of the county funds. The Michigan birth registration law of 1905, originally with no provision for fee, was amended in 1907 to give a compensation of fifty cents to the physician or midwife for reporting each birth. The Kentucky act of 1909 provides a fee of twenty-five cents to physician or midwife for births and of twenty-five cents to the physician for making out a certificate of cause of death. So far no provisions for fees to the undertakers, upon whom the chief burden of filing certificates of death is imposed, has been provided in any registration law. Some of the authorities in charge of the most progressive state systems believe that a fee to the physician or midwife for birth or death registration is not only unnecessary but harmful, so far as securing complete returns is concerned. Physicians may suppose, in a given case of neglect to register a birth, that if the fee is not paid the omission is excused. The payment of a fee would have no detrimental influence, and might perhaps with some individuals smooth the course of registration, provided it were understood, and the law expressly stated, that the failure to receive compensation on account of delayed or incomplete returns should not interfere with the prosecution of every such case and the recovery of the penalty provided by the law.

The work of a registration office, in addition to the responsibility for the thorough and uniform enforcement of the registration law and the proper preservation, binding, and indexing of the records, is really a continuous statistical investigation and should be conducted throughout with direct reference to the statistical data which it is purposed to show. The chief processes of such an investigation may be summarized as follows:

(1) Planning the investigation, which includes the preparation of the schedules or forms used for the "returns"—the technical term defining the filled out blanks or reports which are received at the statistical office as the result of the inquiry.

(2) Scrutiny and correction of the returns. The thoroughness with which this is done obviously depends upon the time at the disposal of the registrar and the accessibility and reliability of the persons reporting.

(3) Hand or mechanical compilation. "Compilation" may comprehend the whole process of obtaining "statistics" from raw material, but is here restricted to the technical procedure of transferring the facts

reported upon the schedules to the "result slips" or "compilation blanks," either directly or through the means of cards.

(4) Tabulation or the making of statistical tables in the form in which they are to appear in the printed reports. This is the only form in which statistics ordinarily are seen by the persons who make use of statistical reports, and it is the chief object of working statisticians to make such tables clear and unmistakable in their form and as comprehensive as possible under the limitations of time, cost, and space.

(5) Finally, an analysis, more or less detailed, by the compiler of the statistics may be of value, as he is presumably best qualified to point out their purpose, the conditions, and qualifications that must be considered in using them, and to indicate generally to the reader the relations between them and similar data for the same or other localities.

Revised United States Standard Certificate of Death.—The certificate of death constitutes the basic schedule for the collection of mortality statistics, and upon the uniformity and precision with which the several items are filled out depends largely the comparability of the resulting data. Prior to 1902 there was the greatest diversity in this respect among the various registration states and cities. A standard form was recommended in that year by the committee of the American Public Health Association, approved by the United States Census, which employed it for all transcripts of deaths received from registration sources, and came into very extensive use by state and city offices. In 1909 this form was revised at the meeting of the American Public Health Association at Richmond and the blank now known as the Revised United States Standard Certificate of Death was recommended for general adoption beginning January 1, 1910. The blank as now in use, together with the standard instructions, which may be supplemented by local offices, intended to secure more precise returns with respect to cause of death and occupation, is given on pages 885 and 886. As reproduced, it is somewhat changed in size. Copies of the certificate can be obtained by request from the Bureau of Census, Washington, D. C.

The twenty items upon the revised U. S. standard certificate of death may be divided into two classes: (1) the personal items, and (2) the statistical items. The latter in their various combinations constitute the material out of which the mortality statistics are created. For convenience they are indicated by italics in the following list:

(1) *Place of Death (Locality).*—This includes identification of the country, state, county, city, or other minor subdivisions of a state, and the wards, sanitary sections, blocks, or other subdivisions of a city.

(2) Full name.—For purposes of identification only.

(3) *Sex.*—This is a primary distinction which ought to be carried out with respect to nearly all statistical compilations.

(4) *Color or Race.*—This is of fundamental importance when the

proportion of colored population is large (as much as 10 per cent.), and demands separate tables or subdivisions of all tables with respect to color when it reaches a higher limit (say 25 per cent.).

(5) *Conjugal or Civil Condition.*—Of little value except when taken in connection with age distribution. Unrelated tables of deaths by conjugal condition are worthless.

(6) Date of Birth.—This may be considered as confirmatory of the statement of age and contributing to the accuracy of the latter statement. When so used it is negligible as a statistical item. It may, however, be selected as the basis of Life Tables, according to certain methods of their construction, in which case it becomes a statistical item of special importance.

(7) *Age.*—Perhaps the most important statistical item after specification of the year and place of death. Exact statement of ages of very young decedents may be essential for the separation of stillbirths from deaths. Valuable tables for public health purposes could be constructed for an area at a given time if the ages at death were the only data afforded; and all comparisons that do not take age distribution into consideration are liable to be misleading.

(8) *Occupation.*—A correct statement, both of the particular kind of work and of the industry, is necessary for satisfactory statistics of occupational mortality—the most important and the most difficult to obtain of any mortality statistics.

(9) *Birthplace (nativity).*—Of special interest in the United States, where the large proportion of foreign-born makes the analysis of mortality by nationality, or at least by general nativity, of importance.

(10) Name of Father.—Of personal and legal importance only.

(11) *Birthplace of father (nativity of father).*

(12) Maiden Name of Mother.—A personal particular of special value for genealogical purposes.

(13) *Birthplace of mother (nativity of mother).*

Items 11 and 13, sometimes in combination as *Birthplace of Parents,* are useful in studying infant and child mortality.

(14) Statement of Informant.—Valuable only as attesting the knowledge of the person filling out the blank as to the personal particulars.

(15) Signature of Registrar and Date of Filing.—Of administrative value only; in this respect important in order to show that the law requiring permits in advance of disposition of the body has been duly complied with.

(16) *Date of Death.*—Statistical reports are usually for calendar years, and hence the statement of year is of primary importance. For monthly or weekly bulletins of vital statistics the basis of compilation may be either the deaths that actually occurred in the period included or

N. B.—Every item of information should be carefully supplied. AGE should be stated EXACTLY. PHYSICIANS should state CAUSE OF DEATH in plain terms, so that it may be properly classified. Exact statement of OCCUPATION is very important. See instructions on back of certificate.

58

885

1 PLACE OF DEATH

County.....

Township.....
or
Village.....
or
City..... (No. ....., ... St.; ..... Ward)

BUREAU OF THE CENSUS

STANDARD CERTIFICATE OF DEATH

State of.....

Registered No.....

[If death occurred in a hospital or institution, give its NAME instead of street and number.]

2 FULL NAME.....

PERSONAL AND STATISTICAL PARTICULARS

3 SEX	4 COLOR OR RACE	5 SINGLE, MARRIED, WIDOWED, OR DIVORCED (Write the word)

6 DATE OF BIRTH
..... (Month) ..... (Day) ....., 1..... (Year)

7 AGE
.....yrs. .....mos. .....ds.

If LESS than 1 day, ...hrs. or .....min.?

8 OCCUPATION
(a) Trade, profession, or particular kind of work.....
(b) General nature of industry, business, or establishment in which employed (or employer).....

9 BIRTHPLACE
(State or country)

PARENTS

10 NAME OF FATHER

11 BIRTHPLACE OF FATHER
(State or country)

12 MAIDEN NAME OF MOTHER

13 BIRTHPLACE OF MOTHER
(State or country)

14 THE ABOVE IS TRUE TO THE BEST OF MY KNOWLEDGE
(Informant).....
(Address).....

15
Filed....., 191.....
REGISTRAR

MEDICAL CERTIFICATE OF DEATH

16 DATE OF DEATH
....., 191.....
(Month) (Day) (Year)

17 **I HEREBY CERTIFY,** That I attended deceased from
....., 191...., to....., 191.....,
that I last saw h..... alive on....., 191.....,
and that death occurred, on the date stated above, at.....m.

The CAUSE OF DEATH* was as follows:

.....
.....
.....
.....(Duration).....yrs.....mos.....ds.

Contributory
(SECONDARY) .....
.....(Duration).....yrs.....mos.....ds.

(Signed)....., M. D.
....., 191..... (Address).....

* State the DISEASE CAUSING DEATH, or, in deaths from VIOLENT CAUSES, state (1) MEANS OF INJURY; and (2) whether ACCIDENTAL, SUICIDAL, or HOMICIDAL.

18 LENGTH OF RESIDENCE (For Hospitals, Institutions, Transients, or Recent Residents)
At place of death.....yrs. .....mos. .....ds. In the State.....yrs. .....mos. .....ds.
Where was disease contracted, if not at place of death?.....
Former or usual residence.....

19 PLACE OF BURIAL OR REMOVAL | DATE OF BURIAL .....191....

20 UNDERTAKER | ADDRESS

REVISED UNITED STATES STANDARD CERTIFICATE OF DEATH

Approved by U. S. Census and American Public Health Association]

STATEMENT OF OCCUPATION.—Precise statement of occupation is very important, so that the relative healthfulness of various pursuits can be known. The question applies to each and every person, irrespective of age. For many occupations a single word or term on the first line will be sufficient, e. g., *Farmer* or *Planter*, *Physician*, *Compositor*, *Architect*, *Locomotive engineer*, *Civil engineer*, *Stationary fireman*, etc. But in many cases, especially in industrial employments, it is necessary to know (*a*) the kind of work and also (*b*) the nature of the business or industry, and therefore an additional line is provided for the latter statement; it should be used only when needed. As examples: (*a*) *Spinner*, (*b*) *Cotton mill;* (*a*) *Salesman*, (*b*) *Grocery;* (*a*) *Foreman*, (*b*) *Automobile factory*. The material worked on may form part of the second statement. Never return "Laborer," "Foreman," "Manager," "Dealer," etc., without more precise specification, as *Day laborer*, *Farm laborer*, *Laborer—Coal mine*, etc. Women at home, who are engaged in the duties of the household only (not paid *Housekeepers* who receive a definite salary), may be entered as *Housewife*, *Housework*, or *At home*, and children, not gainfully employed, as *At school* or *At home*. Care should be taken to report specifically the occupations of persons engaged in domestic service for wages, as *Servant*, *Cook*, *Housemaid*, etc. If the occupation has been changed or given up on account of the DISEASE CAUSING DEATH, state occupation at beginning of illness. If retired from business, that fact may be indicated thus: *Farmer (retired, 6 yrs.)*. For persons who have no occupation whatever, write *None*.

STATEMENT OF CAUSE OF DEATH.—Name, FIRST, the DISEASE CAUSING DEATH (the primary affection with respect to time and causation), using always the same accepted term for the same disease. Examples: *Cerebrospinal fever* (the only definite synonym is "Epidemic cerebrospinal *fever* (never report "Typhoid pneumonia"); *Lobar pneumonia; Bronchopneumonia* ("Pneumonia," unqualified, is indefinite); *Tuberculosis of lungs, meninges, peritoneum*, etc., *Carcinoma, Sarcoma*, etc., of.....(name origin; "Cancer" is less definite; avoid use of "Tumor" for malignant neoplasms); *Measles; Whooping-cough; Chronic valvular heart disease; Chronic interstitial nephritis*, etc. The contributory (secondary or intercurrent) affection need not be stated unless important. Example: *Measles* (disease causing death), *29 ds.; Bronchopneumonia* (secondary), *10 ds*. Never report mere symptoms or terminal conditions, such as "Asthenia," "Anemia" (merely symptomatic), "Atrophy," "Collapse," "Coma," "Convulsions," "Debility" ("Congenital," "Senile," etc.), "Dropsy," "Exhaustion," "Heart failure," "Hemorrhage," "Inanition," "Marasmus," "Old age," "Shock," "Uremia," "Weakness," etc., when a definite disease can be ascertained as the cause. Always qualify all diseases resulting from childbirth or miscarriage as "PUERPERAL *septicemia*," "PUERPERAL *peritonitis*," etc. State cause for which surgical operation was undertaken. For VIOLENT DEATHS state MEANS OF INJURY and qualify as ACCIDENTAL, SUICIDAL, or HOMICIDAL, or as *probably* such, if impossible to determine definitely. Examples: *Accidental drowning; Struck by railway train—accident; Revolver wound of head—homicide; Poisoned by carbolic acid—probably suicide*. The nature of the injury, as fracture of skull, and consequences (e. g., *sepsis, tetanus*) may be stated under the head of "Contributory." (Recommendations on statement of cause of death approved by Committee on Nomenclature of the American Medical Association.)

NOTE.—Individual offices may add to above list of undesirable terms and refuse to accept certificates containing them. Thus the form in use in New York City states: "Certificates will be returned for additional information which give any of the following diseases, without explanation, as the sole cause of death: Abortion, cellulitis, childbirth, convulsions, hemorrhage, gangrene, gastritis, erysipelas, meningitis, miscarriage, necrosis, peritonitis, phlebitis, pyemia, septicemia, tetanus." But general adoption of the minimum list suggested will

those that were reported therein; in the latter case the fact that reported deaths are employed should be stated in order to explain any possible discrepancy between the figures thus given and thus presented in the annual reports based upon the deaths that occurred.

(17) *Cause of Death.*—The most important item from a sanitary standpoint and one that is most difficult to obtain with precision. The subdivision of this item into (a) *primary* and (b) *secondary* introduces special difficulties into the interpretation of the returns prior to the process of "classification" according to the schedule of the International List of Causes of Death, and yet is very necessary in order to give a correct idea of the true causation.

(18) Length of Residence.—Not at present employed as a basis of segregating deaths of non-residents or recent residents. It is desirable that this should be done, to a certain extent, at least in the way of forming supplementary statements to be used in connection with the regular tables that include all deaths that occur in the given area. Precise rules will be necessary for this purpose, and as deaths of residents occurring in other localities cannot, as a rule, be added, statistics of mortality based solely upon deaths of residents will be understatements.

(19) Place and date of burial.

(20) Name and address of undertaker.

There are thus eleven separate statistical items or elements to be considered with reference to each death, or, taking the parent nativity in conjunction instead of that of each parent separately, there are ten statistical elements to be considered in the compilation of deaths. For a given area and unit of time the facts concerning the other eight statistical elements might be shown independently according to appropriate schedules for each. The schedule for sex is simply the statement of males and females, or, in some cases, of unknown sex. That of color, for the United States, usually includes a statement of white, black (or negro), Chinese, Japanese, and Indian. The schedule for civil condition relates to the number returned as single or never married, married, widowed, divorced, and unknown. The schedule for occupations might consist of a simple alphabetical list of occupations returned or a complete "classification," which may embrace occupations or kinds of work in the ordinary sense and also the nature of the industries. The schedule for cause of death is a most complicated and difficult subject, although the purpose is simple, namely, that of giving a list of definite diseases or other causes from which deaths occur.

It is evident that great variety and complexity may occur in the construction of statistical tables relating to deaths, and examination of current registration reports will indicate many practical difficulties in obtaining comparable figures. There is now a movement in the direction of greater uniformity in the preparation of tables, with per-

haps the recommendation of certain standard tables to be presented in all reports, and it is hoped that the result will be greater simplicity, precision, and consequently greater usefulness of the statistical compilations.

Population.—Knowledge of population is back of all intelligent study of vital statistics. Indeed, the study of vital statistics or the registration returns of births, marriages, and deaths is very important for the light that it throws upon the movement of population. The related subjects are included in the general scope of *Demography,* a term not yet in familiar English use.

We may consider population as it exists at a particular time or as it changes according to the observed laws of growth. The first point of view is that afforded by the census. This is taken on a certain day, or, as is the practice in the United States, *as of* a certain day, and presents a cross-section of the conditions then existing. Comparisons of the data of censuses taken at regular intervals may give a correct idea of the general movement of population, but cannot go into the detailed changes that occur from year to year.

Vital statistics alone, that is to say, the data as to the occurrence of certain numbers of births, deaths, and marriages in a given country, state, or city during a certain time, are of little significance unless linked to a statement of the size and character of the population from which they were derived. For the same area and under the tacit assumption that the population has remained substantially constant for a short period of time, valuable comparisons may be made of the absolute numbers of the returns for successive weeks, months, or for a few years. When the period is extended, however, such comparisons cease to be of value and it is necessary to introduce the basic element of population. This is done by the computation of *vital rates.*

Vital Rates.—The comparison of vital events, or of persons affected by vital events, is usually made with the living population by means of *rates* based upon groups containing a definite number of individuals. As an example, the table for England and Wales is selected from the International Tables presented in a recent report (1909) of the Registrar-General of England:

ENGLAND AND WALES—POPULATION, MARRIAGES, BIRTHS, AND DEATHS, 1881-1909

Year	Numbers				Proportion per 1,000 of the Population			Deaths of Children Under 1 Year to 1,000 Births
	Estimated Population in the Middle of Each Year	Persons Married	Births Exclusive of Still-born	Deaths Exclusive of Still-born	Persons Married	Births	Deaths	
1881*	26,046,142	394,580	883,642	491,935	15.1	33.9	18.9	130
1882	26,334,942	408,810	889,014	516,654	15.5	33.8	19.6	141
1883	26,626,949	412,768	890,722	522,997	15.5	33.5	19.6	137
1884	26,922,192	408,602	906,750	530,828	15.1	33.6	19.7	147
1885	27,220,706	395,490	894,270	522,750	14.5	32.9	19.2	138
1886	27,522,532	392,142	903,760	537,276	14.2	32.8	19.5	149
1887	27,827,706	401,036	886,331	530,758	14.4	31.9	19.1	145
1888	28,136,258	407,642	879,868	510,971	14.4	31.2	18.1	136
1889	28,448,239	427,730	885,944	518,353	15.0	31.1	18.2	144
1890	28,763,673	466,056	869,937	562,248	15.5	30.2	19.5	151
1891*	29,085,819	453,052	914,157	587,925	15.6	31.4	20.2	149
1892	29,421,392	454,270	897,957	559,684	15.4	30.4	19.0	148
1893	29,760,842	437,378	914,572	569,958	14.7	30.7	19.2	159
1894	30,104,201	452,898	890,289	498,827	15.0	29.6	16.6	137
1895	30,451,528	456,408	922,291	568,997	15.0	30.3	18.7	161
1896	30,802,858	485,528	915,331	526,727	15.7	29.6	17.1	148
1897	31,158,245	498,290	921,683	541,487	16.0	29.6	17.4	156
1898	31,517,725	510,758	923,165	552,141	16.2	29.3	17.5	160
1899	31,881,365	524,668	928,646	581,799	16.5	29.1	18.2	163
1900	32,249,187	514,960	927,062	587,830	16.0	28.7	18.2	154
1901*	32,621,263	518,800	929,807	551,585	15.9	28.5	16.9	151
1902	32,997,626	523,500	940,509	535,538	15.9	28.5	16.2	133
1903	33,378,338	522,206	948,271	514,628	15.6	28.4	15.4	132
1904	33,763,434	515,712	945,389	549,784	15.2	27.9	16.2	145
1905	34,152,977	521,484	929,293	520,031	15.3	27.2	15.2	128
1906	34,547,016	540,076	935,081	531,281	15.6	27.1	15.4	132
1907	34,945,600	552,842	918,042	524,221	15.8	26.3	15.0	118
1908	35,348,780	529,880	940,383	520,456	14.9	26.5	14.7	120
1909	35,756,615	521,088	914,472	518,003	14.6	25.6	14.5	109

* Census year.

The columns headed "Proportion per 1,000 of the Population" contain the death rates. These are crude, general, or gross annual rates. All rates are taken as *annual* unless otherwise specified, that is to say, they relate to the vital events in the given unit of area (e. g., England and Wales) for the unit of time (one year). If the events are for greater or less periods of time than one year they should be reduced to the form of annual rates for comparison.

The rates are called crude, general, or gross rates because they are based upon the population as a whole, without making allowance for the effect of differences in age and sex distribution. Such rates are the ordinary rates presented in all American registration reports, and form the first step in the comparative study of the vital statistics of different areas. The following tables show the crude birth rates, death rates, marriage rates, and rates of natural increase of population for certain foreign countries for recent years and periods of years:

International Vital Statistics

(From the Seventy-second Annual Report of the Registrar-General of England, 1909)

Country	Quinquennial Periods					Years			
	1881-1885	1886-1890	1891-1895	1896-1900	1901-1905	1906	1907	1908	1909
Persons Married per 1,000 Persons Living									
Austria	15.8	15.5	15.8	16.2	15.7	15.8	15.0	15.3	15.1
Belgium	13.7	14.2	15.1	16.6	16.1	16.1	16.0	15.6	15.3
Bulgaria	17.9	17.4	16.4	16.6	19.8	19.1	19.8	17.7	18.3
England and Wales	15.2	14.7	15.1	16.1	15.6	15.6	15.8	14.9	14.6
France	15.0	14.4	15.0	15.1	15.3	15.6	16.0	16.1	15.7
Germany	15.4	15.8	15.9	16.8	16.1	16.3	16.3	15.9	15.5
Holland	14.3	14.0	14.5	14.9	14.9	14.9	15.0	14.4	14.1
Hungary	20.4	17.8	18.0	17.0	17.2	17.4	19.6	18.2	17.0
Italy	16.1	15.5	14.8	14.3	14.7	15.5	15.4	16.6	15.4
Japan	.....	16.6	17.0	18.1	16.3	14.6	17.7	18.7	17.6
New South Wales	17.0	14.9	13.3	13.9	14.7	15.3	15.7	15.9	16.1
New Zealand	13.6	12.0	12.2	14.2	16.3	17.0	18.0	18.0	16.7
Sweden	12.8	12.2	11.5	12.2	11.8	12.3	12.4	12.2	11.9
Crude Birth Rates per 1,000 Persons Living									
Austria	38.2	37.8	37.4	37.3	35.6	34.9	33.8	33.6	33.4
Belgium	30.7	29.3	28.9	28.9	27.7	25.7	25.3	24.9	23.7
Bulgaria	37.2	35.9	37.5	41.0	40.6	44.0	43.6	40.4	40.6
England and Wales	33.5	31.4	30.5	29.3	28.1	27.1	26.3	26.5	25.6
France	24.7	23.1	22.3	21.9	21.2	20.6	19.7	20.2	19.6
Germany	37.0	36.5	36.3	36.0	34.3	33.1	32.3	32.1	31.1
Holland	34.8	33.6	32.9	32.1	31.5	30.4	30.0	29.7	29.1
Hungary	44.6	43.7	41.7	39.4	37.2	36.0	36.0	36.3	37.0
Italy	38.0	37.5	36.0	34.0	32.6	31.9	31.5	33.4	32.4
Japan	.....	28.5	28.6	31.1	31.7	28.9	33.0	33.9	34.2
New South Wales	37.7	36.4	32.9	28.0	26.7	27.0	27.1	26.8	26.9
New Zealand	36.3	31.2	27.7	25.7	26.6	27.1	27.3	27.4	27.3
Sweden	29.4	28.8	27.4	26.9	26.1	25.7	25.5	24.6	24.6
United States[1]	.....	.....	.....	.....	.....	.....	.....	25.7	25.0
Crude Death Rates per 1,000 Persons Living									
Austria	30.1	28.9	27.9	25.6	24.2	22.4	22.6	22.5	22.9
Belgium	20.6	20.2	20.1	18.1	17.0	16.4	15.7	16.5	15.8
Bulgaria	17.7	18.9	27.8	23.9	22.5	22.3	22.3	24.3	26.6
England and Wales	19.4	18.9	18.7	17.7	16.0	15.4	15.0	14.7	14.5
France	22.2	22.0	22.3	20.7	19.6	19.9	20.2	19.0	19.3
Germany	25.3	24.4	23.3	21.2	19.9	18.2	18.0	18.1	17.2
Holland	21.4	20.5	19.6	17.2	16.0	14.8	14.6	15.0	13.7
Hungary	33.1	32.1	31.8	27.9	26.2	24.8	25.2	24.8	25.1
Italy	27.3	27.2	25.5	22.9	21.9	20.8	20.7	22.6[2]	21.5
Japan	.....	20.6	21.1	20.7	20.9	19.8	20.9	21.0	22.0
New South Wales	15.7	13.8	12.8	11.9	11.2	9.9	10.6	10.1	9.6
New Zealand	10.9	9.9	10.1	9.6	9.9	9.3	10.9	9.5	9.2
Sweden	17.5	16.4	16.6	16.1	15.5	14.4	14.6	14.9	13.7
United States (registration area only)	.....	.....	.....	.....	16.2	15.7	16.0	14.8	14.4
Natural Increase (Excess of Births over Deaths) per 1,000 Persons Living									
Austria	8.1	8.9	9.5	11.7	11.4	12.5	11.2	11.2	10.5
Belgium	10.1	9.1	8.8	10.8	10.7	9.3	9.6	8.4	7.9
Bulgaria	19.5	17.0	9.7	17.1	18.1	21.7	21.3	16.1	14.0
England and Wales	14.1	12.5	11.8	11.6	12.1	11.7	11.3	11.8	11.1
France	2.5	1.1	0.0	1.2	1.6	0.7	0.5	1.2	0.3
Germany	11.7	12.1	13.0	14.8	14.4	14.9	14.3	14.0	13.9
Holland	13.4	13.1	13.3	14.9	15.5	15.6	15.4	14.4	15.4
Hungary	11.5	11.6	9.9	11.5	11.0	11.2	10.8	11.5	11.9
Italy	10.7	10.3	10.5	11.1	10.7	11.1	10.8	10.8	10.9
Japan	.....	7.9	7.5	10.4	10.8	9.1	12.1	12.9	12.2
New South Wales	22.0	22.6	20.1	16.1	15.5	17.1	16.5	16.7	17.3
New Zealand	25.4	21.3	17.6	16.1	16.7	17.8	16.4	17.9	18.1
Sweden	11.9	12.4	10.8	10.8	10.6	11.3	10.9	10.8	11.9

[1] For provisional registration area for births only; this was established for the first time for 1908 and is much smaller than the registration area for deaths.

[2] Including 77,283 deaths in the earthquake at Messina and Reggio (Dec. 28, 1908); excluding these deaths the death rate was 20.3.

The data for the United States have been added to the rates for the countries given in the International Statistics, and relate only to deaths in the registration area since 1900 and to births in the provisional registration area established for 1908. The latter is much smaller than the death registration area, the returns are not yet fully tested—it is possible that certain areas included do not really record as high a proportion as nine out of every ten births that occur—and hence no comparison of the births and deaths can be made for the establishment of the rate of natural increase of population as shown for the other countries.

To proceed with the examination of the table containing the vital statistics of England and Wales, it will be observed that the careful qualification is made for both births and deaths that the statements given are "Exclusive of stillbirths." This follows as a matter of course in England, where stillbirths are not registered, except under local provisions, but in many countries, and especially in the United States, it is extremely important to know, in examining the statistics for a series of years, whether stillbirths were or were not counted among the births and among the deaths. The present practice is to exclude them from both, so that the term "Births" means living births or children born alive, and "Deaths" means the deaths of those only who were counted among the living births. Of course in taking the difference of births and deaths, either both inclusive or both exclusive of stillbirths, the amount of natural increase is not affected.

Persons married, or twice the number of marriages, is usually preferred to the number of marriages per 1,000, although the latter rate is frequently employed. The comparison is more direct with the persons forming the population.

Lastly, the basis of all the rates (except the rate of infantile mortality in the last column) is the column showing the estimated population in the middle of each year. The English census is taken about April 1 in the years designated by an asterisk in the table. Censuses of various countries vary greatly in the precise period of the year in which they are taken, hence it is customary to prepare midyear estimates, even for census years, upon which to base the vital rates. Of course, all intercensal populations must be estimated, and also postcensal populations, or populations of years following the year of the last census available. The years 1902-1909 are postcensal years, but as soon as the census of 1911 was taken new estimates of population can be interpolated for the years between 1901 and 1911, which thus become intercensal years as considered with reference to the last enumeration of population.

The computation of crude vital rates is very simple, the practical rule being to annex three ciphers to the number of births (exclusive of still-

births), deaths (exclusive of stillbirths), stillbirths, and persons married (twice the number of marriages), and divide by the estimated midyear population. This is for rates per 1,000 of mean population, and rates per 1,000 are the generally accepted bases of comparison and usually to be understood when the word *rate* is used, except when individual causes are referred to. It is unnecessary to carry the computation beyond two places of decimals nor to express the result further than to the first decimal place, although it is not unusual to express the second decimal figure. It adds nothing to precision to carry the quotient out at great length. The death rate of the registration area of the United States in 1910 was 15.0 per 1,000, as derived from a midyear population of 53,843,896 persons and 805,412 deaths. This is just as accurate as the statement of 14.96 or 14.958 per 1,000. It is customary in the United States to express death rates of individual causes as rates per 100,000 population, chiefly to avoid the use of fractional expressions. Thus a death rate from typhoid fever of 41.9 per 100,000 seems more significant than when stated as a rate of 0.42 per 1,000. Of course, the two expressions are quite identical, as also those of 4.2 per 10,000, and 419 per 1,000,000.

The variety of ratios employed for the expression of mortality rates is needlessly large, and it would be desirable to limit the number. As an example the provisionally estimated population of England and Wales for the year 1909 was 35,756,615, the total number of deaths from all causes was 518,003, and the number of deaths from diphtheria was 5,235. The relations between the population and deaths may be stated in several ways:

Expressed as	*All Causes*	*Diphtheria*
Death rate per million	14,487.	146.
Death rate per hundred thousand	1,448.7	14.6
Death rate per ten thousand	144.87	1.46
Death rate per thousand	14.487	0.146
Death rate per hundred (per cent.)	1.4487	0.0146
Death rate per ten (never used)	0.14487	0.00146
Death rate per unit	0.014487	0.000146

If the rates were always carried out as above it would be easy to convert one form of expression into any other by shifting the position of the decimal point as required. The death rate per 1,000 from all causes would ordinarily be written as 14.5 or 14.49. The Registrar-General's reports have for many years given the rates for individual causes as per 1,000,000, without fraction, a form with which the rate per 100,000, with fraction, as used in the American reports, is readily comparable by dropping the decimal. Recently, however, some English tables have employed the rate per 1,000 for individual causes (e. g., diphtheria, 0.15), a method which has the advantage of bringing the rate

for each disease more closely into touch with the rate for all causes. It is not necessary to refer to the reciprocal expressions obtained by dividing the population by the deaths, since the use of these is practically obsolete in registration reports. They have the disadvantage that changes are not readily comparable and that the numbers increase as the mortality diminishes.

All rates ordinarily employed in registration reports are central death rates, as distinguished by the actuary from rates of mortality based upon the deaths and population entering upon an age or period, except the rate of infantile mortality, which is the number of deaths of infants under one year of age per 1,000 births, not per 1,000 population under one year of age.

All rates are also customarily stated as annual rates, although for use in weekly or monthly bulletins they may be computed upon returns that include only a portion of the year. The assumption is made that *if* the number of deaths that occurred during the week or month continued with the same frequency for the year the annual rate would be equal to the product of the number of deaths observed in the period into the ratio between the period and the year, divided by the mean population for the year. Such rates are ephemeral and usually subject to the limitations incident to the use of small numbers. It should be quite sufficient to multiply the deaths in a week, especially if the reported and not the actual deaths, by 52, inasmuch as the assumption of a fixed mean population introduces an artificial fluctuation into the series of weekly rates. A more exact relation between the week and the year is given by the multiplier 52.17747; by dividing the estimated mean population of the year by this number a working weekly mean population is obtained by which the number of deaths in the week is divided directly to give the weekly rate in annual form. For months the multipliers may be 11.8, 12.2, and 13 for 31-day, 30-day, and 28-day months, respectively. Closer approximations may be used, a mean monthly population can be computed for each month, and the computation of rates for individual causes may be facilitated by finding the rate for one death and multiplying by the number of deaths from each cause. Too much refinement is unnecessary, as the monthly rates are subject to a considerable systematic error unless graduated monthly estimates of population are used instead of the estimated population in the middle of the year.

Methods of Estimating Population.—There is no way of knowing the exact population of a country, state, or city except by actual enumeration, and this can be done only for the year of the census. It is understood, of course, that there is a certain margin of error in all census enumerations, no matter how carefully conducted. This is not, as a rule, sufficient to be of any consequence, but in the statement of

population the figures below thousands have no significance for large areas in which millions of population are involved. It would be quite as correct to take the mean population to the nearest thousand for England and Wales in 1901 (32,621,000) as to give the exact number, based upon the method of computation applied to the census taken on April 1, 1901 (32,527,843), which resulted in a midyear estimate of 32,621,263. Indeed, it is likely that even the thousands are uncertain, and as there may also be some understatement of vital events, especially of births in various countries, rates are not always entitled to absolute credence to the ultimate places of decimals sometimes employed. All vital rates based upon estimated populations (and practically all rates are in fact so based) and upon registration less than absolutely complete are to be considered as approximate expressions, and not as perfect statements of truth. The actual errors, as a rule, are probably slight, counterbalancing (as in the slight understatement, both of population and vital events), and may be neglected for practical purposes.

Since it is not feasible to attempt to fix exactly the population for any year lying between two censuses or following the latest census, the purpose of estimating populations for non-census years is to afford a reasonable and uniform basis whereby the vital rates of a country and its subdivisions may be compared. Laying aside the method of keeping an exact account of the excess of births over deaths and the gain or loss from migration, which is not generally practicable, the chief methods employed for this purpose are those known as the (1) geometrical and the (2) arithmetical methods.

The geometrical method assumes a constant *rate* of increase between census years or, in other words, that the amount of annual increase is in proportion to the population. This assumption would be absolutely correct for a population dependent entirely upon natural increase for its growth and in which the birth rates and death rates remained constant or varied in the same direction and by the same amount, so that their difference or the rate of natural increase for each year was the same. Thus, with an initial population of 100,000 persons, an initial birth rate of 30 per 1,000, less a death rate of 20 per 1,000, would afford a rate of natural increase of 10 per 1,000 (or 1 per cent.) per annum. The amount of increase would be 1,000 persons and the population of the second year would be 101,000. With the same rate of increase the number added for the next year would be 1,010, not 1,000, as for the year before; the additional 10 persons represent the rate of growth applied to the increment of the previous year. In other words, it is a simple application of the principle of compound interest and, the rate of growth remaining constant, the annual increments of population are a little larger each year.

The arithmetical method assumes a constant *amount* of increase be-

tween census years. The total increase of population from one census to the next is divided by the number of years between the censuses, and the resulting average annual increase is added from year to year to give the estimated populations. For example, if a certain population increased from 100,000 persons in 1900 to 110,000 persons in 1910, the annual increment of population, according to this method, would be 1,000 and the estimated population for 1901 would be 101,000 persons; for 1902 it would be 102,000 persons, and so on. It is evident that as the same number of persons is added each year to a constantly increasing basis of population the annual *rate* of increase is a decreasing one.

The choice of methods for a given area, aside from considerations of uniformity, may depend upon whether the population is in fact increasing at a uniform rate or one approximately so. If the annual rates of increase are uniform the decennial rates will also be uniform and the geometrical method will be the method of choice. For example, if a state showed an increase of population amounting to 20 per cent. from 1890 to 1900 and a like increase of 20 per cent. from 1900 to 1910 it is reasonable to suppose that the annual rates of increase are the same for the two decades ($\sqrt[10]{1.20} = 1.0184$, or 1.84 per cent.). With an initial population of 100,000 in 1890 the population of 1900 would be 120,000, and that of 1910 would be 144,000. Estimates for the postcensal years following 1910 might be made on the assumption that the rate found constant, or practically so, for the preceding decades would continue as the rule of growth. But suppose the population increased only as much from 1900 to 1910 (20,000) as it did from 1890 to 1900. It is evident that the rate of growth has decreased, and it would lead to error to base estimates upon the hypothesis of a constant rate when the decennial increments plainly indicate a diminishing rate. The use of equal amounts of annual increase is more suitable for populations for which the decennial periods do not show fairly constant or increasing percentages of growth.

A practical illustration of the method of computation of postcensal populations according to each method may be made for England and Wales for the years 1902-1909. The estimates are based on the populations given for the middle of the preceding census years 1891 and 1901,[1] on the following page.

The arithmetical method is the more easily applied. The difference between the populations of 1891 and 1892 is the actual increase of population during the decade, and one-tenth of the difference represents the average annual increase (353,544.4) which is added successively to give the estimated populations of the postcensal years. The fractional part is carried only for convenience, so that the result for 1911 will check exactly with the sum of the population of 1901 and the decennial

[1] See table on p. 896.

Year	Geometrical Method		Arithmetical Method
	Number	*Logarithm*	*Number*
1901	32,621,263	7.5135008	32,261,263
1891	29,085,819	7.4636813	29,085,819
Difference		0.0498195	3,535,444
One-tenth of difference		0.0049819.5	353,544.4
1901	(32,621,263)	7.5135008	32,621,263
Add		0.0049819.5	353,544.4
1902	(32,997,626)	7.5184827.5	32,974,807.4
Add		0.0049819.5	353,544.4
1903	(33,378,338)	7.5234647	33,328,351.8
Add		0.0049819.5	353,544.4
1904	(33,763,434)	7.5284466.5	33,681,896.2
Add		0.0049819.5	353,544.4
1905	(34,152,977)	7.5334286	34,035,440.6
Add		0.0049819.5	353,544.4
1906	(34,547,016)	7.5384105.5	34,388,985
Add		0.0049819.5	353,544.4
1907	(34,945,600)	7.5433925	34,742,529.4
Add		0.0049819.5	353,544.4
1908	(35,348,780)	7.5483744.5	35,096,073.8
Add		0.0049819.5	353,544.4
1909	(35,756,615)	7.5533564	35,449,618.2
Add		0.0049819.5	353,544.4
1910	(36,169,150)	7.5583383.5	35,803,162.6
Add		0.0049819.5	353,544.4
1911	(36,586,454)	7.5633203	36,156,707
Check:			
1901	(32,621,263)	7.5135008	32,621,263
Add (difference, 1891-1901)		0.0498195	3,535,444
1911		7.5633203	36,156,707

increase. In reading the estimated population for each year the fraction is disregarded if less than 0.4 and added as an additional unit if 0.5 or greater.

The geometrical method deals with the logarithms, and not with the natural numbers. The difference is the logarithm of the *ratio* of

decennial increase of population from 1891 to 1901, and is equivalent to dividing 32,621,263 by 29,085,819, or 1.1215, which may be otherwise stated as an increase of 12.15 per cent. during the decade. One-tenth of the difference of the logarithms is the logarithm of the rate of annual increase which, applied year by year in the manner of compound interest, would afford the ascertained rate of decennial increase. It is equivalent to extracting the tenth root of the ratio of decennial increase, and in the present computation amounts to 1.0115, or, otherwise stated, 1.15 per cent. The estimated populations (antilogarithms) for each year are taken from the tables of logarithms to correspond to the logarithms found by the successive addition of the one-tenth difference to the logarithm of the population of the last census year, and the result is checked by agreement with the sum of this logarithm and that of the difference at the end of the decade. For ordinary purposes and for smaller populations it is not necessary to use seven-place tables; in fact, four-place tables are sufficiently accurate for most uses of vital statistics.

The final test of a series of estimated populations for postcensal years lies in its agreement with the results of the following census. All postcensal estimates are provisional in character, and should be revised as soon as the latest census gives a new fixed point by which the true nature of the change in population can be determined. The results of the English census of 1911 are now available and the estimated population for the middle of the year 1911 as given in the Quarterly Return of Marriages, Births, and Deaths, published by the Registrar-General for the second quarter of that year, was 36,168,750, or 417,704 less than the population estimated according to the rate of increase between 1891 and 1901. The excess of the estimated over the actual population was only 1.15 per cent., a proportion so small when we consider the long period between the censuses that it confirms the general value of the rates computed for the intercensal years. The difference between the actual and the arithmetically estimated population was even less (—12,043, or —0.33 per cent.). It happens that the arithmetical method would have given better results for the past decade in this particular instance. The geometrical method is the one generally employed by European statisticians, and conformity to international usage is an important point in its favor. The arithmetical method has been used by the Bureau of the Census for the registration area of the United States since 1900, after a comparative study which showed that it was more applicable to the observed conditions of growth of American populations. The results of the last census indicate some areas, however, in which a more uniform rate of growth from decade to decade suggests that the geometrical rate of increase is becoming more consistent with conditions of growth in the United States.

For the United States at present it is desirable that all populations employed in state and city registration reports and bulletins should be estimated according to the arithmetical method, so that the figures will be comparable upon a uniform basis and in agreement with those employed by the Federal census. A resolution to this effect was adopted by the American Public Health Association. Where the geometrical method is preferred, an alternative series of rates should be presented. The populations estimated by the latter method are usually greater for post-censal years, so that a very considerable reduction in the apparent rates may occur at the end of the period. For intercensal estimates, on the contrary, the populations by the arithmetical method are greater, so that the rates on the basis of the geometrical estimate are higher, especially for the middle years of the period. The arithmetical method has also the great practical advantage that estimates made for the subdivisions and the whole of an area exactly balance. It is necessary to use it even when the general principle of the increase in geometrical progression is accepted. Following is the statement of the method actually employed in the Registrar-General's reports:

(1) The populations of the whole country and of its various constituent portions are calculated on the assumption of a rate of increase in arithmetical progression equal in each case to that obtaining in the previous intercensal period. The sum of the populations so calculated for parts of the country equals that of the whole. [This is the United States Census method.]

(2) The population of the whole country is recalculated on the assumption of a rate of increase in geometrical progression equal to that obtaining in the preceding intercensal period.

(3) The relation between the arithmetical and geometrical results so obtained for the whole country is expressed by the factor resulting from the division of the geometrical progression estimate by the arithmetical.

(4) The estimates for portions of the country, based upon the assumption of continued increase in arithmetical progression, are multiplied by the above factor. The sum of the results equals the estimate for the whole country by geometrical progression obtained in stage 2.

In the preceding comparison of the arithmetical and geometrical methods of estimating population as applied to England and Wales the basic populations were the estimated midyear populations of 1891 and 1901. These midyear populations were themselves estimated by the geometric method from the census figures which relate to a date about the beginning of the second quarter of the year. In the United States, as the dates of the censuses were nearly in the middle of the year (as of June 1) until that of 1910, no allowance has heretofore been made for the difference between the date of enumeration and the middle of

the year. Beginning with 1910, however, such an allowance has been made in the Census mortality statistics, and the intercensal populations for the preceding intercensal periods have been recomputed on this basis, without, however, disturbing established rates as of June 1 for census years prior to 1910, whether state or federal. The method employed is explained in the *Annual Bulletin on Mortality Statistics*, 1909, the example taken being the District of Columbia (city of Washington):

Year and Exact Date of Census or Estimate	Method I Present Estimates Based on Censuses of June 1, 1890, and June 1, 1900	Method II Revised Estimates Based on Census of June 1, 1900, and Estimate for June 1, 1910	Method III Revised Estimates Based on Censuses of June 1, 1900, and April 15, 1910	Method IV Revised Estimates Based on Census of June 1, 1900, and Estimate for July 1, 1910	Method V Revised Estimates Based on Estimates for July 1, 1900, and July 1, 1910
1890 (June 1)	***230,392**				
1891	235,225				
1892	240,057				
1893	244,890				
1894	249,722				
1895	254,555				
1896	259,388				
1897	264,220				
1898	269,053				
1899	273,885				
1900 June 1	***278,718**	***278,718**	***278,718**	***278,718**	
1900 July 1, estimated					*279,160
1901	283,551	284,019	283,953	284,064	284,461
1902	288,383	289,321	289,188	289,409	289,763
1903	293,216	294,622	294,423	294,755	295,064
1904	298,048	299,924	299,658	300,100	300,365
1905	302,881	305,225	304,894	305,446	305,667
1906	307,714	310,526	310,129	310,791	310,968
1907	312,546	315,828	315,364	316,137	316,269
1908	317,379	321,129	320,599	321,482	321,570
1909	322,211	326,431	325,834	326,828	326,872
1910 April 15			***331,069**		
1910 June 1, estimated	327,044	*331,732			
1910 July 1, estimated				*332,173	*332,173
1911		337,033	336,304	337,519	337,474
1912		342,335	341,539	342,864	342,776
1913		347,636	346,774	348,210	348,077
1914		352,938	352,009	353,555	353,378
1915		358,239	357,245	358,901	358,680
1916		363,540	362,480	364,246	363,981
1917		368,842	367,715	369,592	369,282
1918		374,143	372,950	374,937	374,583
1919		379,445	378,185	380,283	379,885
1920 March 1, estimated			383,420		
1920 June 1, estimated	375,370	384,746			
1920 July 1, estimated				385,628	385,186

"In the above table census figures are printed in boldfaced type and the average annual increase in each column is taken between the figures distinguished by asterisks as the bases for the estimated populations.

"Method I represents the method of estimating population used up to the present time. The enumerated populations for the census years 1890 and 1900, both taken as of June 1, are used as the bases. The difference between these populations, divided by 10, represents the average annual increase from June 1, 1890, to June 1, 1900. This

difference, added successively to each of the years 1891 to 1899, gives the estimated population for each year of the *intercensal* period, and the continued addition of the same annual average to the years 1901 to 1909 would give the *postcensal* estimates on the same basis, that for the year 1910 (June 1) being 327,044, or only 4,025 less than the actual enumerated population (April 15), 331,069. The postcensal estimates by this method have been used in the annual reports for 1901 to 1908, and there would have been little objection to using the estimate for 1909 in this individual case, but for some areas the difference might have been considerable and the intercensal estimate for 1909, based upon the census of 1900 and 1910, is more desirable.

"Method II shows how the estimates as of June 1 might be supplied for the intercensal period 1900 to 1910 and continued for future years by estimating the population as of June 1 from the enumeration of April 15, 1910, and comparing as usual with the former census of June 1, 1900.

"Method III is an objectionable one, but is shown because registration officials may attempt perhaps to make estimates by direct comparison of the population as enumerated June 1, 1900, and April 15, 1910, without regarding the differences in dates of enumeration. While it is true that in many cities population resident on April 15 is more nearly the correct midyear population than would be the population actually enumerated as of July 1, it is certain that for the country as a whole considerable growth takes place between April 15 and the middle of the year. Consequently, if we compare an enumeration of June 1, 1900, with an enumeration not ten years later, but less than ten years later by one and one-half months, it is evident that the estimated population on this basis ten years subsequent, or for 1920, would be for three months prior to June 1, or as of March 1.

"Method IV. Another method would be to obtain the midyear estimate for 1910 and compare it directly with the enumeration of June 1, 1900, disregarding the differences in dates for the intercensal period 1900 to 1910, then continuing with midyear estimates. This would be fairly satisfactory, although it would seem preferable to adopt the plan next in order.

"Method V, which has been adopted by the Bureau of the Census for the intercensal estimates from 1900, or from the date of the latest state census, to 1910, and for future estimates of population beginning with 1911, is the midyear estimate, or that of approximately July 1. It makes necessary a slight computation before using the actual results of enumeration according to the census of April 15, 1910, but the estimated midyear populations for 1900 and 1910 once having been obtained, the subsequent interpolations or extrapolations are very simple. No change is proposed in the arithmetic method, or that of the average

annual increase, which has given good results for the past decade and is more applicable to the observed method of growth of population of this country than the geometric method.

"The process of estimating the midyear populations for 1900 and 1910 from the census enumerations as of June 1 and April 15, respectively, is very simple. In former estimates, when each census was of date June 1, the interval between them was exactly ten years, or 120 months. The interval between the census of June 1, 1900, and the census of April 15, 1910, is not 120 months, but only 118.5 months; dividing the observed increase of population for a given area by 118.5, the average monthly increase during the decade is obtained. This monthly increase added to the population June 1, 1900, gives the midyear population for 1900, and two and one-half times the monthly increase added to the population of April 15, 1910, gives the midyear population for 1910. One-tenth of the difference between the two midyear populations is then added successively for the intercensal years 1900 to 1910 and the postcensal years beginning with 1911. Suitable allowance must, of course, be made for changes of area."

Specific and Corrected Death Rates.—The death rates heretofore considered have been based upon the comparison of total deaths or the deaths due to particular causes with the aggregate population. Each element of the population has its peculiar relation to the rate of mortality, and therefore for exact comparisons the constitution of the population must be considered. The death rate of males per 1,000 male population is practically always higher than the death rate of females per 1,000 female population; for England and Wales, 1909, the rates were 15.4 and 13.7, respectively, the ratio of male to female mortality being 112 per cent. Hence a state or country with a relatively greater proportion of female population than another will normally show a lower death rate. The female death rate, in the march toward lower mortality which has been the characteristic of the vital statistics of the past few decades, seems to lead the way and to be followed after an interval by the male death rate.

Color or race, especially in the United States, is a very important element of population. The total death rate of the registration area for the census year 1900 being 17.8 per 1,000, the death rate of the white population included was 17.3, and that of the colored population was 29.6. But the colored population was entirely urban, so that the comparison is somewhat unfair, because the death rates of cities are usually found to be higher than of the country. Much of the difference between the death rates of the white and colored in various cities may be due to conditions of housing, employment, or other factors not dependent at all upon essential differences in the race relation to disease. The death rates of various nationalities vary greatly. The death rates of cities having

large proportions of foreign-born population living under insanitary conditions, in crowded tenements, or otherwise exposed to unfavorable conditions, are higher than those of cities more favorably situated. It is unfair to compare the death rates of the white population alone in certain cities with the gross death rates in other cities without considering that the colored mortality omitted corresponds to the mortality of a laboring and tenement class not otherwise represented. It is desirable, however, that the deaths of the colored should be segregated from those of the white, with distinction as to whether negro, Indian, Chinese, or Japanese, whenever they form a considerable proportion of total deaths, and that separate tables by sex and age be shown therefor.

One of the most important factors to be considered in comparing the death rates of various areas is the distribution of population by age. The following table shows, for the group of registration states as constituted in 1900, the percentages of population and deaths at each decennial age group from birth to the end of life, and also the corresponding data for the first two quinquennial periods of age and for the first year of life, with the *specific death rates* for each age:

Age	Population		Deaths		Death Rate per 1,000 Population of the Same Age
	Number	Per Cent.	Number	Per Cent.	
All ages	19,960,742	100.0	343,217	100.0	17.2
Under 1 year	437,944	2.2	71,117	20.7	162.4
Under 5 years	2,072,797	10.4	103,529	30.2	49.9
5 to 9 years	1,984,486	9.9	9,242	2.7	4.7
Under 10 years	4,057,643	20.3	112,771	32.9	27.8
10 to 19 years	3,624,065	18.2	14,169	4.1	3.9
20 to 29 years	3,745,605	18.8	27,546	8.0	7.4
30 to 39 years	3,104,747	15.6	28,322	8.3	9.1
40 to 49 years	2,261,283	11.3	27,152	7.9	12.0
50 to 59 years	1,558,210	7.8	30,987	9.0	19.9
60 to 69 years	977,227	4.9	37,556	10.9	38.4
70 to 79 years	464,260	2.3	38,631	11.3	83.2
80 to 89 years	118,651	0.6	21,471	6.3	181.0
90 years and over	9,908	0.0	3,424	1.0	345.6
Unknown age	39,143	0.2	1,188	0.3	30.4

The corrected death rate known as the "Index of mortality," which was adopted by the International Statistical Institute in 1895 on the basis of recommendations made by Dr. Josef von Körösi, considers only the proportions of the population at five groups of ages, and applies the specific death rates at these ages as ascertained for each country or state to the population of Sweden at the census of 1890, the distribution of which at these age groups per 1,000 is known as the "Standard population." As an example, the computation of the index of mortality accord-

ing to this method for New South Wales is taken from the Official Year-Book of the Commonwealth of Australia, 1911:

New South Wales Age Groups	Mean Population, 1909, Distributed According to Results of Census of 1901	Number of Deaths 1909	Number of Deaths per 1,000 of Mean Population, 1909, in Each Age Group	Age Distribution per 1,000 of Standard Population	Index of Mortality
Persons (all ages).....	1,605,863	15,810	9.85	1,000.0	13.44
Under 1 year.....	40,484	3,234	79.88	25.5	2.04
1 to 19 years.....	704,050	1,960	2.78	398.0	1.11
20 to 39 years.....	514,932	2,251	4.37	269.6	1.18
40 to 59 years.....	256,586	2,965	11.56	192.3	2.22
60 years and over.....	89,811	5,400	60.13	114.6	6.89

The crude death rate is found by dividing the total number of deaths in the year (15,810) by the estimated midyear population (1,605,863), and amounts to 9.85 per 1,000. When, however, the deaths at the five age-groups are divided by the estimated midyear population of each age-group the specific death rates are obtained for each group. The result of such specific rates for a population having the constitution of New South Wales is indicated by the crude rate already obtained (9.85), but what would the rate have been if applied to other populations of greater or less favorable age constitution? It is impracticable to compare New South Wales with every other country or state individually, but the same object is attained by reducing New South Wales and any other state or country to a basis of comparison by means of a selected population, which happened to be that of Sweden at the last census prior to the date of the establishment of the system. So a typical thousand by age is taken, out of which 25.5 were infants under one year of age. The ratio to total population is small, but the death rate (not infant mortality) of this group is high (79.88 per 1,000 = nearly 8 per cent.); consider it as applied to a population of only 25.5 persons and as a result or contribution to the mortality of the typical thousand 2.04 deaths would occur at this age. (It is unfortunate to have to speak of fractional deaths in this connection, but we are considering a thousand typical of a much larger actual population.) The next group, 1 to 19 years, has the lowest death rate, but the largest proportion of the standard population. Three hundred and ninety-eight persons, with a death rate of only 2.78 per 1,000, would afford only 1.11 deaths to the mortality of the typical thousand. And so on, noting that the high death rate at sixty years and over (60.13), affecting over 11 per cent. (114.6 per 1,000) of the standard group, contributes over half of the total deaths (6.89). For the total deaths of the standard thousand are of course

obtained by adding together the deaths that would have occurred at each age period *if* the several specific death rates at those periods had been such as actually occurred in New South Wales during the year 1909. This corrected death rate or mortality index (13.44) exceeds the crude rate by 3.59 per 1,000, or nearly four points of mortality, and shows that the age distribution of New South Wales was favorable to that extent as compared to the age distribution of the population of Sweden in 1890. And so the rates of the other states and the Commonwealth of Australia would be raised, for the same year and under the same assumption of identical age distributions in 1909 and 1901, as follows: Victoria, crude rate 11.24, corrected rate 13.74; Queensland, 9.79 to 13.80; South Australia, 9.37 to 12.57; Western Australia, 9.93 to 15.07; Tasmania, 10.00 to 13.14; and the Commonwealth as a whole from 10.22 to 13.56. Comparisons with other countries are not given in the report and do not seem to have been made very generally according to this system.

Any other country, the average of a group of countries, or even an arbitrary age distribution, might have been selected as a basis of comparison. In point of fact the most valuable corrections of mortality data for both age and sex, together with the most extensive list of international comparisons, have been made in the annual reports of the Registrar-General of England, in which for some years past death rates corrected according to the age and sex distribution of England and Wales in 1901 have been presented for the leading countries of the world, together with tables of specific death rates and of the distribution of the population of each country by sex and age at recent censuses. The results for the aggregate of both sexes, together with corresponding crude and corrected rates for the registration states of 1900, are shown in the table on page 905.

It will be observed that for the group of registration states as constituted in 1900 the change from the crude to the corrected figure is but a slight one, and the actual rate is practically the same as that of England and Wales. Some of the states, as, for example, Maine, have a less favorable age and sex constitution of the population than that of the average, as a result of which the rate obtained by applying the specific death rates to the standard population is considerably less than the crude rate. The death rate of Sweden corrected on the English standard population shows a decrease from 16.78 to 13.88, or nearly 3 per 1,000. The death rate of New South Wales corrected on the English standard rose from 11.72 to 13.10, a difference of 1.38, while the correction for the year 1909 upon the Swedish population of 1890 as a standard shows an increase from 9.85 to 13.44, or 3.59. On the whole the English standard would seem most convenient for use in the United States, as involving less change in the size of the rates, but

MEAN ANNUAL CRUDE AND CORRECTED DEATH RATES[1] PER 1,000 LIVING IN ENGLAND AND WALES AND IN CERTAIN EUROPEAN COUNTRIES AND BRITISH COLONIES.

Countries (Arranged in Order of Their Corrected Death Rates—Persons)	Deaths to 1,000 Living	
	Persons	
	Corrected Death Rates	Crude Death Rates
Russia (European) (1896–8)	28.61	32.80
Spain (1900–02)	26.53	27.63
Hungary (1899–01)	24.87	26.34
Austria (1899–01)	23.12	24.83
Bulgaria (1899–01)	20.92	23.26
Italy (1900–02)	20.23	22.72
Prussia (1899–01)	19.70	21.08
German Empire (1901)	19.52	20.84
Finland (1899–01)	19.12	20.54
Scotland (1900–02)	17.61	17.91
France (1900–02)	17.50	20.80
England and Wales (1900–02)	17.16	17.16
Switzerland (1899–01)	16.86	18.22
Belgium (1899–01)	16.78	18.53
Ireland (1900–02)	16.59	18.27
Western Australia (1900–02)	15.83	13.72
The Netherlands (1898–1900)	15.40	17.32
Sweden (1899–01)	13.88	16.78
Denmark (1900–02)	13.63	15.80
Queensland (1900–02)	13.29	11.89
New South Wales (1900–02)	13.10	11.72
Victoria (1900–02)	13.08	13.12
South Australia (1900–02)	11.73	11.02
Tasmania (1900–02)	11.44	10.88
New Zealand (1900–02)	10.80	10.01
United States (registration states, 1900)[2]	17.04	17.19
Connecticut	17.37	18.02
District of Columbia	24.36	22.25
Indiana	14.37	14.19
Maine	14.85	17.11
Massachusetts	18.13	18.25
Michigan	13.90	14.03
New Hampshire	16.31	18.55
New Jersey	18.20	17.75
New York	18.26	18.21
Rhode Island	20.87	20.45
Vermont	13.80	16.56

[1] The corrected death rates are the death rates at all ages that would have resulted from the rates prevailing at the various age groups if the sex and age constitution of the populations in the several countries had been identical with that of the population of England and Wales as enumerated at the Census of 1901.

[2] Data for the United States added to the original table.

some method of correction should be employed, especially for the comparison of the death rates of cities with widely different age constitution of population, as otherwise the most misleading inferences as to comparative healthfulness may be drawn.

Classification of Causes of Death.—A large part of the work of a registration office consists of the tabulation of the causes of death, and it is necessary, for the sake of uniformity and comparability of the re-

sulting statistics, that this shall be done according to certain recognized methods. The selection of a uniform classification is the first essential, and it is fortunate that we are now in possession of a system which is in practical use in a large number of countries. The International Classification of Causes of Death, sometimes known as the "Bertillon System," from the name of the distinguished demographer, Dr. Jacques Bertillon, who reported it to the International Statistical Institute at Chicago in 1893, has been employed by the U. S. Bureau of the Census since 1900, is used by all the registration states, and has been adopted by Great Britain beginning with 1911. The complete list of countries using it may be found in the report of the International Commission of Revision, which meets every ten years for the purpose of revising it so that it may be abreast of medical progress. The last revision was made in Paris in 1909. A Manual of the International List has been published by the Census Office and also a Physicians' Pocket Reference, which has been distributed to all the physicians of the United States so that they may aid in the precise statement of causes of death and so improve the quality of the mortality statistics in this respect. The Bellevue Hospital Nomenclature of Diseases and Conditions has also been arranged in the order of the International List, so that it is now available for use by hospitals for recording and statistical purposes.

The use of a classification, or statistical list, as it is preferably called, is a process of condensation of the multitudinous terms employed by physicians upon certificates of death to a uniform list of fairly precise titles. It is unnecessary to present the International List in full in this work because it is readily available in the official statistical reports and other publications issued by the Federal and state authorities and by England (after 1911) and other countries. As an example the "inclusions" for typhoid fever, the first title of the tabular list, may be shown as presented in the Census *Manual* (1911):

I—GENERAL DISEASES.

1. Typhoid Fever

This title includes:
Abdominal fever
typhoid
typhus
Abortive typhoid
Ambulant typhoid
Cerebral typhoid
typhus
Continued fever
Enteric fever
Enterica
Gastroenteric fever
Hemorrhagic typhoid fever
Ileotyphus
Intermittent typhoid fever
Malignant typhoid fever
Mountain fever
Paratyphoid fever
Paratyphus
Posttyphoid abscess
Rheumatic typhoid fever
Typhobilious fever
Typhoenteritis
Typhogastric fever

1. Typhoid Fever *Continued*

This title includes:

Typhoid fever
malaria
meningitis
stupor
ulcer
Typhomalaria
Typhomalarial fever
Typhoperitonitis
Typhus (unqualified)[1]
abdominalis

It is unfortunate that physicians, in reporting causes of death, should not write "typhoid fever" invariably, and no other expression, when typhoid fever is in fact the cause of death. The difficulty is even greater with respect to many other causes, as, for example, acute anterior poliomyelitis and cerebrospinal fever (epidemic cerebrospinal meningitis). The Physicians' Pocket Reference contains a list of indefinite and undesirable terms, framed by the Committee on Nomenclature of the American Medical Association in conjunction with the Government services concerned, and there is a prospect of a further reform in practical medical nomenclature. The selection of the cause of death for statistical tabulation when two or more causes are returned upon the certificates is important. Variations in this respect may affect the resulting statistics to an appreciable degree. Some of the principles employed are indicated in the following suggestions for the preference of jointly returned causes of death as given in the Manual of the International List of Causes of Death:

"For returns upon the Standard Certificate of Death, and especially for those returns in which the instructions have been regarded by the reporting physicians, the following suggestions for classifying may be helpful:

"1. Select the primary cause, that is, the real or underlying *cause of death*. This is usually:

"(a) The cause first in order.

"(b) The cause of longer duration. If the physician writes the cause of shorter duration first, inquiry may be made whether it is not a mere symptom, complication, or terminal condition.

"(c) The cause of which the contributory (secondary) cause is a frequent complication. See lists of 'Frequent complications' under the various titles of the Tabular List.

"(d) The physician may indicate the relation of the causes by words, although this is a departure from the way in which the blank was intended to be filled out. For example, 'Bronchopneumonia *following*

[1] The majority of deaths returned in the United States from "typhus" or "typhus fever" are in reality from typhoid fever. Deaths properly chargeable to International Title No. 2 (Typhus fever) are so infrequent in this country that the Bureau of the Census invariably makes an effort to identify each as a case of true exanthematic typhus. If no additional information can be obtained, "typhus" is compiled under (1) and "typhus fever" under (2).

measles' (primary cause last) or 'Measles *followed by* bronchopneumonia' (primary cause first).

"2. If the relation of primary and secondary is not clear, prefer general diseases, and especially dangerous infective or epidemic diseases, to local diseases.

"3. Prefer severe or usually fatal diseases to mild diseases.

"4. Disregard ill-defined causes (Class XIV), and also indefinite and ill-defined terms (e. g., 'debility,' 'atrophy') in Classes XI and XII that are referred, for certain ages, to Class XIV, as compared with definite causes. Neglect mere modes of death (failure of heart or respiration) and terminal symptoms or conditions (e. g., hypostatic congestion of lungs).

"5. Select homicide and suicide in preference to any consequences, and severe accidental injuries, sufficient in themselves to cause death, to all ordinary consequences. Tetanus is preferred to any accidental injury, and erysipelas, septicemia, pyemia, peritonitis, etc., are preferred to less serious accidental injuries. Prefer definite means of accidental injury (e. g., railway accident, explosion in coal mine, etc.) to vague statements or statement of the nature of the injury only (e. g., accident, fracture of skull).

"6. Physical diseases (e. g., tuberculosis of lungs, diabetes) are preferred to mental diseases as causes of death (e. g., manic depressive psychosis), but general paralysis of the insane is a preferred term.

"7. Prefer puerperal causes except when a serious disease (e. g., cancer, chronic Bright's disease) was the independent cause.

"8. Disregard indefinite terms and titles generally in favor of definite terms and titles."

References to Sources and General Precautions in Use of Statistical Data.—All figures are not statistics and, as a rule, for serious statistical studies reference should be made to the original sources of the data as found usually in the official reports. Every sanitary and registration official, as well as all students of comparative statistics, should have access to the annual reports of the Registrar-General of England on births, deaths, and marriages. Besides affording examples of the extensive use of corrected death rates for a population with vital statistics very similar to those of the United States and probably more generally comparable to those of this country—if we possessed them in as full detail—than those of any other nation, the Registrar-General's reports present each year valuable tables of international vital statistics prepared through the coöperation of the official registrars of practically all countries with adequate registration. The United States appeared in this collection for the first time in 1909, with tables, of course, relating only to the registration area and for deaths alone. Another publication of the Registrar-General, the "Annual Summary of Marriages, Births,

and Deaths in England and Wales, and in London," gives death rates of many cities of the world from certain important diseases, the rates of infantile mortality, etc.

For a general view of the vital statistics of the world the *Statistique Internationale du Mouvement de la Population, 1905*, published by the French Government in 1907 under the direction of M. Lucien March, Director of the General Statistical Bureau of France, is the most convenient and reliable source of reference. Even though the extremely interesting text should not be fully available to English-speaking persons, the figures themselves, most carefully verified and reduced from official reports, are in that universal language independent of the limits of nationality that constitutes the body of statistics. Uniformly estimated populations afford the bases of vital rates, which may be traced from their beginnings in Sweden (1749) and Finland (1750) until the present time, when practically all civilized countries save the United States are represented. The data are brought down, as far as possible, to 1905, so that rates for the first quinquennial period of the century (1901-1905) are available, and presumably will be continued as soon as the official reports for the following quinquennium are at hand. In this connection a word may be said relative to the foolish preference sometimes expressed for the "latest data." Vital statistics carefully recorded and tabulated are an imperishable possession, and for many purposes observations made several years before the date of use are just as cogent as the results of the latest year. It is not necessary that statistics should be "news" to be useful; and, in fact, the careful preparation of certain kinds of vital statistics requires that the reports should be deferred until the material has been carefully digested. For example, the Supplement to the Sixty-fifth Annual Report of the Registrar-General of England (1902), containing Life Tables and statistics of occupational mortality based upon the returns of deaths for 1891-1900, was not published until 1907-08; the magnificent German Life Tables (Kaiserlichen Statistischen Amte) for the same period appeared in 1910. Of course, annual reports may be issued within a reasonable time after the year to which they relate, but even for these, as a rule, more time in preparation and condensation rather than hasty publication of crude and inaccurate data would be desirable. For immediate use monthly and weekly bulletins may give all essential information required.

An especially valuable compilation for city registration officials is the *Statistique Démographique des Grandes Villes du Monde, 1880-1909*, published for the Thirteenth Session of the International Statistical Institute at The Hague, 1911, by the Municipal Bureau of Statistics of Amsterdam. The first part contains data for European cities, and is to be followed by a volume containing data for other great cities of the world. The tables relate to the movement of population, with rates

for persons married, births, deaths, and natural increase of population based upon populations estimated to the middle of each year, births and stillbirths by legitimacy and sex, deaths of infants under one year per 100 live births of the year preceding, deaths under one year (including stillbirths) per 100 total births (including stillbirths) of the same year, and deaths and death rates per 100,000 population from scarlet fever, diphtheria (including croup), whooping-cough, typhoid fever, pulmonary phthisis, and cancer.

For the mortality statistics of the United States recourse will first be had to the annual reports published by the Bureau of the Census. That for 1909 presented revised rates for all causes and individual causes of death for all areas (registration states, including cities of 10,000 population and over, rural population of counties exclusive of such cities, and registration cities in non-registration states) for each year of the decennial period 1900-1909. Annual reports are also published by various states and cities, and in some of these are presented standard tables containing estimated populations and rates for preceding years. It is always well to examine the series of estimates for assurance that they have been made in some regular and systematic manner, which should always be mentioned in connection with the table, as rates based upon mere guesses or reckless estimates, and not revised and corrected after the following census has proved them to be incorrect, may be found in certain reports. In general, for the United States any reports or bulletins that present rates based upon less than the total number of deaths that occurred in the area for the time covered should be discarded. Some offices exclude deaths of non-residents according to the arbitrary interpretation of this term by the local official, and without including deaths of residents that may occur elsewhere, deaths from violence, deaths of infants under one day or under one week old, etc., and yet venture to compare their vitiated rates with those regularly computed by the majority of American offices in accordance with the Rules of Statistical Practice adopted by the American Public Health Association. The reckless haste to produce a low death rate (in mere figures, not facts) has led to all sorts of unjustifiable tampering with statistical returns.

SECTION X

INDUSTRIAL HYGIENE AND DISEASES OF OCCUPATION

Industrial hygiene is one of the most important topics in preventive medicine, as it deals with the health, the welfare, and the human rights of the vast majority of the population. Industrial hygiene is a subject in which the medical, economic, and sociologic aspects are closely interwoven, and it requires a broad grasp and intimate knowledge of the conditions to avoid the dangers and correct the injustices to which workpeople are subjected. The questions of industrial hygiene strike at the very root of our social fabric; they include the relation of capital and labor, and the relation of man to his fellow men. The man of means may, to a large extent, select not only the place, but even the character of his employment. He can choose his own hours of work and can largely control his environment while at work, so far as it affects his health and comfort; he can purchase fresh air, sunshine, good food, rest, recreation, and other conditions that make for health, longevity, and happiness. The employee must largely accept the conditions as he finds them and is frequently denied many advantages, even necessities. As the power of the employee is limited, he needs the assistance of the state to correct the unreasonable demands which capital has ever exacted of labor. Legislators should champion the rights of work-people, especially in the realm of industrial hygiene. Our country has been negligent in this regard and has fallen far behind England and continental countries. The situation has received some assistance through organized labor, which has exerted a good influence in limiting the avarice of the employer, in shortening the hours of work, in obtaining a more just share of the profits, in improving sanitary conditions, and in exacting a modicum of human consideration. Thus when the stone masons came to build the Hygienic Laboratory in Washington they refused to work until a proper shelter and other reasonable conveniences were provided, as required by their labor union.

Modern conditions have brought entirely new problems into industrial hygiene. These have come about largely through the development of new industries and the invention of new processes, through improved and changed methods of transportation, and through specialization and

crowding in cities and work places, through artificial light, through changing relations between capital and labor, and the intensive and unrelenting pressure of the times. Some of the conditions which oppress the workmen are brought about by the greed of capital and disregard of the human machine, but indifference, carelessness, and ignorance of the workman himself are responsible for many avoidable accidents and preventable maladies. In Eastman's study of work accidents in Pittsburgh it appeared that, out of 410 fatal accidents, the victim or his fellow workers were responsible in 188 cases and the employer in 147 cases. Despite the improvements in labor-saving devices the human machine will ever remain the most vital and indispensable machine in the production of wealth—at the same time it is the most delicate and sensitive machine. Both from the standpoint of humanity and the standpoint of economy the human machine deserves greater care and consideration than any other mechanism engaged in the production of wealth.

There are especial dangers to health incident to certain industries, such as liability to lead poisoning in the manufacture of white lead; of phossy jaw in the manufacture of matches made with white phosphorus; of caisson disease in divers and those who work in compressed air; there are extra hazards to life and limb in railroading, mining, and among those who work with explosives; there is a particular danger to those who are compelled to work in a dusty atmosphere, more so if the dust is of an irritating or poisonous nature; and there is danger to those who are compelled to breathe poisonous fumes such as carbon monoxid, hydrogen, sulphid, mercury, etc. These special instances represent the true diseases of occupation. There are many other influences, not specifically inherent to industry, to which the workman is often subjected which seriously influence health, such as poor ventilation, lack of cleanliness, overcrowding, excessive hours, improper light, fatigue, and a hundred and one conditions which affect the health and the efficiency of the workman. These examples are usually considered under industrial hygiene. Work should be ennobling, and anything which tends to degrade it is morally wrong.

The statistics of morbidity and mortality in relation to diseases of occupation need careful scrutiny, especially when used for comparison. The factors which enter into such statistics are so numerous and the conditions so variable that misleading conclusions are common. The workmen come and go, they vary very much in physical vigor to start with, are of all ages, both sexes, many nationalities, and are greatly influenced by home conditions and by the character of their recreation. Many industries, while not in themselves particularly hazardous, are rendered so through intemperance or dissipation. The statistician must be careful to take all factors into account that bear upon the subject.

Some industries are blamed for conditions affecting health that really are due to the insanitary home conditions and bad habits of the individual.

In recording the nature of a man's work it is not sufficient simply to state that he is a laborer, mechanic, machinist, mill operator, and the like. Such information is frequently of no more value to the student of the diseases of occupation than the name of the person himself. If the person is a blacksmith or works with heavy metals it is plain that

FIG. 132.—RED OXID OF LEAD AND LITHARGE BEING MIXED IN THE MANUFACTURE OF STORAGE BATTERIES. The workman is wearing a respirator, but should also protect himself with long-wristed gloves.

he works under a severe physical strain. If he is a sailor upon a sailing ship we know that he is exposed to rough weather and unusually severe conditions, whereas if he is a sailor upon a modern passenger steamship the conditions of his work may be no more severe than those of the janitors and charmen in a large office building. If he is in finance we may be sure that he is subject to severe nervous strain. It is therefore not sufficient simply to give the name of the trade, but detailed inquiry should be made into the nature of the work and the particular conditions under which he works.

With the exception of phosphorus, lead, and mercury poisoning, little, if any, investigations of value have been made in this country into the vast question of industrial poisoning. That careful study carried out by competent authorities is urgently needed can be seen from the following list of poisons that are in every-day use in our industries: methyl alcohol, ammonia, anilin, antimony, arsenic, arsenuretted hydrogen gas, benzol, carbon bisulphid, carbon monoxid, chlorid of lime, chlorin, chromium, di-nitro-benzol, formaldehyd, mineral acids, manganese, nitrobenzin, nitrous oxids, picric acid, prussic acid, pyridin, sulphuretted hydrogen, and many more.

Our country has long nursed the delusion that the conditions under which our workmen operate are better than corresponding conditions abroad. Recent investigations have shown the contrary to be the fact. Thus, Dr. Alice Hamilton discovered 25 cases of lead poisoning during one year in a "model" Illinois factory employing 200 hands, while large English white lead works under careful supervision frequently show not a case of lead poisoning for several successive years. We are just waking up to the seriousness of the situation—the first American Congress on Industrial Diseases met in June, 1910.[1]

Andrews estimates that in the United States 30,000 wage earners are killed by industrial accidents every year, and that at least 500,000 more are seriously injured. A memorial on industrial injuries prepared by a committee of inspectors, appointed by the President of the Association for Labor Legislation, states that there are probably not less than 13,000,000 cases of sickness each year among those engaged in industrial employments. The money lost each year (for those who find dollars more expressive than lives) is conservatively calculated at nearly three-fourths of a billion dollars. At least one-fourth of this painful incapacity for work and consequent economic loss can be prevented.

The diseases of occupation refer only to those diseases which are contracted because of the occupation and which would not have been contracted if the individual had not engaged in that particular occupation. Most of the diseases of occupation are due to poisonous and irritating gases, vapors, or dust to which work-people are exposed. Special affections are sometimes caused by exposure to high temperatures, abnormal atmospheric pressures, and other unusual conditions peculiar to some occupations. These are the true occupational diseases. In addition to these there are a large number of affections caused by insanitary factory or office surroundings, such as overcrowding, bad air, imperfect light, lack of cleanliness, improper washing or toilet facilities, etc.

[1] The Massachusetts legislature of 1904 took the first step in this country to obtain definite scientific data on the subject of the occupational diseases. The State Board of Health of Massachusetts made an investigation into the scientific condition of factories, workshops, and mercantile establishments, and published its first report on the subject in 1907.

When disease is contracted or health undermined through such insanitary conditions it cannot be regarded as an inherent or special danger of a particular occupation. If a person contracts typhoid fever or diphtheria through the use of a common drinking cup or a roller towel in a factory, workshop, or office these evidently cannot be considered occupational diseases. On the other hand, if a person contracts lead poisoning because he is required to carry lead bars this is evidently a danger inherent to his occupation, and thus becomes a true disease of occupation. The general sanitary and hygienic conditions under which work is done are comprised under the term "industrial hygiene," while the maladies caused by exposure to poisonous fumes, dust, or other special dangers during a manufacturing process comprise the true diseases of occupation. Industrial hygiene, from the standpoint of the sanitarian, is simply a special application of our general knowledge bearing upon the health and welfare of mankind. Industrial diseases, on the other hand, require a special study as to their causes, symptoms, and modes of prevention.

An industry may be a nuisance or disturbance to the community as well as to those engaged in its various processes. Thus the noise of a tack factory or boiler shop, the smells from glue or fertilizing factories, or the fumes from smelting or chemical works, or the smoke from chimneys or locomotives, wastes from tanneries, paper mills, and mines do not come directly in the chapter of industrial hygiene or the diseases of occupation, however closely related.

SOME FUNDAMENTAL CONSIDERATIONS IN PREVENTION

In order to improve the hygienic conditions under which people work, and in order to prevent the diseases of occupation, five fundamental conditions are essential: (1) investigations; (2) laws; (3) factory inspection; (4) penalties; (5) education. It is self-evident that before anything may be accomplished a careful study must be made of the facts. These investigations must include not only scientific studies, but also economic and sociological factors. Suitable laws are necessary, for it has been found in practice that the conditions cannot be corrected by an appeal to voluntary reform. To be effective the laws must provide ample ways and means for their energetic enforcement. A systematic factory inspection is necessary in order not only to protect workpeople against the preventable diseases of occupation and to correct sanitary defects, but also to enforce the laws concerning hours of occupation, child labor laws, and related subjects. These laws have little force unless they provide a penalty both against the employer and the employees. Either party to the contract should be held legally

responsible in case of violation. Finally, education directed to the employer, the employee, and also to the public at large is necessary to obtain the laws and maintain the standards.

Hours of Work.—No general rule can be laid down for the hours of work, which may vary with the character of the employment. Thus the hours of active work are limited by a smith or glass-blower, a worker in a caisson or mine, a locomotive engineer, and other occupations necessitating great muscular effort or intensive concentration, or exposure to unnatural conditions. Formerly men worked at the quieter occupations all the time not given to sleep; now the day is better divided into eight hours of work, eight hours of "re-creation," and eight hours of sleep. Hygienically, it is important to have one full day's rest each week. It cannot be maintained from the medical side that working longer than eight hours a day is harmful to health, but it is held that no employer has the right to utilize the greater part of a man's day and thus deprive him of the leisure to which he, as a human being, is entitled. Since his whole nature has to be developed, time must be given for the intellectual, moral, and physical welfare of man, which cannot take place if the hours of employment are too long, the work too hard, or of a grinding nature. The hours of work depend somewhat upon the physical exertion required and also upon the nervous tension. The Saturday half holidays, especially during the heated term; a vacation period, and a tendency to increase the number of holidays are all signs of social improvement which make for health and happiness.

Fatigue.—Economic engineers find that it pays to give employees a rest at stated intervals and to guard the conditions surrounding workers, so that they are neither molested nor interrupted, that the light and other factors are agreeable, and the sanitary surroundings good. Work and rest must be judiciously alternated. Efficiency ceases when fatigue begins. The danger to the workman himself, as well as to others, is now recognized from a tired brain, tired nerves, and tired muscles. Accidents are especially prone to happen to workmen who are tired. Thus most accidents in factories happen as the day wears on. The effect of fatigue on the occurrence of accidents is graphically shown by French and Belgian statistics. The number of accidents increases progressively during the morning hours, drops after the noon intermission, and then rises from hour to hour until the end of the working day, affording a practical illustration of Helmholtz's experiments in attention fatigue. Fatigue is not only dangerous to the workman himself, but sometimes to others; thus the overwrought and tired-out train dispatcher may send trains into collision. Further, fatigue of certain nerves and muscles may result in definite symptoms such as writers' cramp, or more general manifestations such as nervous prostration.

Typewriters, telegraph operators, and others suffer from these occupational neuroses.

Next to fatigue, nervous tension and worry are very wearing, and when combined become especially harmful. Diabetes prevails among engine drivers to a considerable extent. Worry, hurry, and a high nervous tension are recognized as a frequent predisposing cause of ill health or breakdown in all walks of life, including the so-called higher professions.

FIG. 133.—AN EFFECTIVE DUST-REMOVING SYSTEM IN THE BOOT-AND-SHOE INDUSTRY. Edge trimming. (Mass. State Board of Health.)

Children.—The first factory act in this country was passed by the state of New York in 1886. By this act no child under 13 years of age was allowed to work in factories. Since then the minimum has been raised to 14. The injustice to the child and the consequence upon its health and development of subjecting it to the monotony and grind of factory life are too evident to need emphasis. Recently it has been claimed that in certain districts, as, for example, the mill district of our southland, the children are better off in a good textile mill of modern construction than they are living under the insanitary conditions of their homes. It would be just as logical to state that they would improve in

health if removed to a prison or almshouse. The child of to-day is the citizen of to-morrow and his health and development are the most important assets of the state. In Germany children under 12 are not allowed to work in factories; between 12 and 14 they are not allowed more than 6 hours per day, and between 14 and 16 not more than 10 hours per day. Further, they are not allowed to begin work earlier than 5.30 a. m. nor work later than 8.30 p. m., and one hour is required for dinner. Children are not allowed at all in certain dangerous trades, as coal mines, etc. In this country child labor is legally prohibited in factories, upon the stage, and other undesirable places in many of the states: the question with us is somewhat complicated on account of the industrial competition between the states. The regulation of child labor and compulsory education are too important for longer delay. There are certain occupations in which minors under no circumstance should be permitted to engage. This includes the dangerous trades in which there is liability to accident and the trades in which there is danger to health on account of irritating dust or poisonous fumes. There are also certain occupations, such as messenger boys at night, which should be entirely prohibited by law on account of the exposure to temptation.

Women.—Women are physiologically not capable of doing the same work as men, especially during the period of maternity. Further, several days each month women are more or less incapacitated for most kinds of work on account of menstruation. Pregnant women should not work for several weeks before labor, and after labor not until the uterus has undergone involution, which is a matter of another month. In Switzerland the law requires a total of 8 weeks before and after labor. This is a wise law which all enlightened countries should accept. Justice demands that women should be given full pay during this time, which is of such great moment to her own health and that of her offspring. Mr. Brandeis successfully defended the constitutionality of the ten-hour law for women in Oregon. The brief submitted by this eminent jurist in a similar action before the Illinois Supreme Court [1] should be read by those interested in this subject. The primary object of this brief is to show that the demands of public health require legal restrictions in the work of women because of the peculiar importance to the community of the health of mothers. The effect of overwork on the different organs is reviewed, also the effect of night work, of prolonged standing on the feet, of foot-power machinery, and of the speeding up required by the "piece-work system." The general literature upon fatigue and overwork is reviewed.

The effect of overwork upon fecundity and upon infant mortality is

[1] Brandeis, Louis D., assisted by Goldmark, Josephine: Brief and Argument for Appellants. In the Supreme Court of the State of Illinois, December term, 1909.

impressive. Broggi states that of 172,365 Italian women between the ages of fifteen and fifty-four years who were employed in industrial occupations the average child-bearing coefficient was only about one-third of the general fertility of Italian women.

It is now a well-established fact that infant mortality is shockingly high among the babies of women who work in factories and mills. It has been shown in Germany and England that infant mortality increases progressively according to the increase in the proportion of women obliged to work outside of their homes, and this is true even if the mother's work results in higher standards of comfort in the home. The two classical demonstrations of this rule are the great Lancashire cotton famine and the Siege of Paris, during both of which crises there were loss of employment and great privation. In spite of the starvation and the increased general death-rate, the infants' death rate fell in Paris actually to 40 per cent. simply because the women, being out of work, were obliged themselves to nurse and care for their children. The infant mortality in industrial centers such as Fall River, Lowell, and Lawrence, in Massachusetts, which are mill towns, is twice as high as similar towns without many factories and no overcrowding.

It is plainly the duty of the nation to restrict the hours of work of women and also prohibit their employment in certain industries known to be particularly hazardous to the sex. Saleswomen should be provided with seats in shops so as to avoid the ill effects of prolonged standing, they should have one or two days each month for rest during the menstrual period, and should be protected against undue strain and fatigue. While women's work may be regulated in the industries and the hours of employment may be limited by law, there can be no law to regulate women's work in the household which is "never done." Men have still to learn the lesson that nervous breakdown and the results of fatigue are as harmful in women who overwork in the home as in those who work in shops and factories. The long hours and confining work of house servants sometimes lead to anemia and other troubles. Cooks are exposed to the effects of excessive heat and to sudden changes of temperature. Domestic "servants" as a class supply a large contingent of patients in hospitals and out-clinics. The long hours and insufficient sleeping accommodations, as well as the nature of the work, lead to ill health which may in part account for the disinclination of women to accept this kind of service.

Factory Inspection.—There is no longer doubt but that factory inspection is necessary as a protection to the workman. An efficient system requires a good comprehensive basic law and a capable corps of inspectors. The inspectors should be thoroughly familiar with the law and with the processes of manufacture and also with the problems of preventive medicine. Factory inspectors should be capable of making

recommendations outside of the strict regulations under which they operate so as to improve conditions and meet the needs of an ever changing situation. Factory inspection really falls into two categories, one of which deals mainly with the medical side and the other with the legal and economic side. Both inspectors should take into account the social and humanitarian side. Some of the factors which should engage the attention of a factory inspector are: ventilation, dust, gases, vapors, odors, temperature, moisture, light, cleanliness, over-crowding, excessive heat, dampness, drinking-water, children, women, washing facilities, water-closets, cloakrooms, receptacles for expectoration, defective sanitary arrangements, hours of work and rest, the age of the employees, their physical condition, etc. Hanson points out that medical men, through their training and attitude, make the best factory inspectors, for they alone are in a position to make the best use of facts, and learn something of the sanitary conditions of premises where men and women work, to study the possible injurious effects of certain processes, to inspect devices designed to protect the employees against injury or against dangerous fumes and dust, and to judge the effects on the health of operatives of such substances, as well as to detect the symptoms of certain poisons incident to such occupations, to detect and protect the employees and others from infectious diseases, to make physical examinations of minors, and to collect and make proper use of all facts and data, including morbidity and mortality statistics, pertaining to occupational hygiene. The medical inspector is also able to correlate the injurious influences in the factory, in the home, and in the habits of the individual.

Preventable Accidents.—The most obvious and striking of the preventable accidents occur on railroads, in mines, and in factories. About 10,000 persons are killed and 100,000 more or less seriously injured on the railroads of the United States every year. Some 3,000 fatal accidents occur annually in the course of mining operations, and probably 5,000 deaths result from accidents, in the operation of machinery in factory and workshop. Much of this is preventable, in fact, prevented in other countries. Winslow points out that fatalities are four times as common among our railroad employees as among those of England, and other accidents seven times as frequent. Coal mining was nearly as fatal in Belgium between 1830 and 1840 as it is in the United States to-day, but the Belgians have cut their death rate down to less than one-third of what it was.

A system of workmen's compensation, by which the victim of industrial accidents, except when caused by his own neglect, is entitled by right, and without legal proceedings, to a proper money equivalent for the injury received, is simple justice which has been long delayed in this country. Workmen's compensation laws have been in successful operation in all the principal European countries. Many of our larger

corporations voluntarily and automatically compensate employees in case of accidents. In 1910 New York, Montana, and Maryland passed laws making such an arrangement optional or compulsory for certain classes of occupations. In New York the law was declared unconstitutional. In 1911, 10 different states, California, Illinois, Kansas, Massachusetts, Nevada, New Hampshire, New Jersey, Ohio, Washington, and Wisconsin, enacted laws bearing on this subject.

FIG. 134.—SYSTEM OF HOODS AND VENTILATORS TO CARRY OFF THE FUMES FROM THE FURNACES IN A FOUNDRY. (Mass. State Board of Health.)

Sedentary Occupations.—Sedentary occupations in themselves may lead to harm, especially in the cases of those who bend forward while at work, causing contraction of the chest and pressure upon vital organs which interferes with important physiological functions. The circulation is impeded, respirations are shallow, the ultilization of food is diminished and the appetite fails, constipation and hemorrhoids are common, and there is a predisposition to common colds and diseases of the lungs.

DISEASES OF OCCUPATION

Classification of the Occupational Diseases.—Oliver divides the occupational diseases into five general classes:

(1) Diseases due to gases, vapors, and high temperatures.

(2) Diseases due to conditions of atmospheric pressure.

(3) Diseases due to metallic poisons, dusts, and fumes.

(4) Diseases due to organic or inorganic dust and heated atmospheres.

(5) Diseases due to fatigue.

Many other classifications have been attempted, but it is evident that no general system can be entirely satisfactory. Each occupation requires individual study and separate consideration. In many occupations a combination of varying factors, such as dust fumes, poisons, fatigue, etc., operate coincidently. In the following pages only the well-known and better studied diseases of occupation and the conditions which render them hazardous, as well as methods of prevention, are considered. The number of occupational diseases is rapidly growing as the subject is receiving more careful attention. Thus recently it has been shown that workers with heated tallow and other animal grease are subject to gastro-intestinal disturbances, apparently due to the volatile fatty acids that are given off and that the workers ingest and inhale. Strumpf and Zable [1] describe chronic antimony poisoning among type-setters. Skin diseases are frequently found among workers in the following: galvanizing, cutters of glass and pearl shell, workers with tar, paraffin, arsenic, cement, dyes, printer's ink, chromium, potassium permanganate, and among polishers.

LEAD

Lead poisoning is one of the most frequent, most serious, and most insidious of all the occupational intoxications. If a pound of lead drops on a workman's head the catastrophe is more obvious than if minute quantities of lead salts are taken into the system day by day, but the poisoning may be as fatal as the accident. The population at large is also frequently poisoned with lead from a variety of sources. Thus the lead may be in the drinking-water as a result of contact with lead pipes, in canned goods from the solder, in foods cooked in lead-enameled utensils, and from handling lead or objects containing lead.

Lead is a typical cumulative poison. A large amount may be taken at one time without noticeable effect, but small quantities ingested daily are absorbed, stored in the body, resulting in chronic poisoning and even death. Lead is excreted both by the kidneys and the liver, and also the skin. It probably does not appear in the urine except with albumin, that is, lead can only pass a damaged kidney. The lead excreted by the liver passes into the intestines with the bile and may be found in the feces. The elimination of the lead, however, is slow and uncertain. As much as one ounce of the acetate of lead has been taken at one time without

[1] *Zeitschr. f. experimentelle Path. und Pharmakologie*, 1910, LXIII, p. 242.

injury. Older physicians frequently prescribed the acetate of lead as an astringent in doses of 10, 20, or 30 grains. The same amount of lead distributed in minute doses and taken daily would, in all likelihood, result in serious poisoning. The reason for this is that when one large dose is taken only a small quantity is absorbed; the rest is swept through the intestines, but when small quantities are taken at frequent intervals practically all is absorbed and the metal accumulates in the tissues, poisoning especially the delicate nervous structures.

The susceptibility to lead poisoning varies greatly. Of a number of persons exposed to the same conditions some are fatally poisoned, others suffer with mild plumbism, and still others escape entirely. Young persons are much more susceptible than old. Young adults suffer most. Women are more susceptible than men. Recognizing this fact, in 1898 England abolished female labor in the dangerous processes of white lead manufacture. The reasons for this varying susceptibility are only partly understood. Hyperacidity of the gastric juice is a predisposing factor, because the lead in such persons is readily converted to the soluble chlorid in the stomach. Personal cleanliness is another important factor, and workers in lead who do not give scrupulous attention to cleanliness of person and clothing suffer most. Persons who are not particularly careful about cleaning their hands before eating, or who frequently carry their fingers to their mouth and nose, run especial risks. Oliver has shown by experiments on animals that alcohol precipitates attacks of plumbism, a fact which, in the human subject, clinical experience has again and again confirmed. There is not the least doubt that alcoholic intemperance predisposes to lead poisoning.

Practically all forms of lead are poisonous, even the metal itself. The carbonate, the oxid, and the chromate are the most serious because these are most employed in the industries. The soluble salts are more readily absorbed than insoluble salts.

In the majority of cases of lead poisoning in the industries the lead comes through the air to the victim as dust, sometimes as fumes. Preventive measures must, therefore, be directed toward keeping the air about the workmen free from lead. A lead trade is dangerous in proportion to its dustiness. Lead is usually taken into the system from the digestive tract, although absorption from the respiratory tract and even through the skin may occur. For many years it was a disputed point whether the lead entered through the skin or the intestinal tract, but it is now conceded that the intestinal mucosa, also that of the mouth, is the usual portal of entry. Much of the lead dust that is a source of lead poisoning is, in fact, swallowed.

The water-soluble salts of lead such as acetate, chlorid, and nitrate may be absorbed through the skin, but this is slow and requires long exposure. It is possible that the non-soluble salts may be changed by

contact with the fatty acids on the skin into soluble compounds. Lead poisoning may be caused by absorption through the skin from cosmetics containing lead. Edsall thinks skin absorption relatively unimportant. In this he is in accord with Weyl, Legge, Oliver, and Sommerfeld.

The symptoms of lead poisoning are: a blue line on the gums (sulphid of lead), a diminution in the secretion of saliva and a sweetish taste in the mouth, colic, constipation, weakness, slowing of the pulse, increase in blood pressure, and anemia. The corpuscles may fall below 50 per cent. and many of the red cells show a granular basophilic degeneration when stained with one of the polychrome methylene blue dyes. Lead palsy is very common. It is a peripheral toxic neuritis and usually affects a localized group of muscles such as the extensor muscles of the forearm—painter's wrist drop. The common symptoms are colic, constipation, and paralysis. Edsall calls attention to the fact that encephalitis, which expresses itself as an acute insanity, is a frequent manifestation of lead poisoning.

The character of certain occupations has an influence on the type of lead-poisoning which develops. Thus Teleky finds that, while compositors in Vienna seldom suffer from colic or from the severer types of lead poisoning, they are subject to an unusual extent to diseases of the lungs and kidneys. The relation between tuberculosis and chronic plumbism is shown in Hahn's diagrams based on the records of typographical trades in Vienna and Berlin, the curves of the two diseases showing a remarkable parallelism. Colic is said by Legge to be most frequent among workers in white lead, red lead, enameling, storage-batteries, coach-painting (which involves sandpapering), while the severer form with paralysis is found in brass-workers, plumbers, printers, file-cutters, and tinsmiths. The former are very dusty trades; poisoning occurs rapidly and encephalopathy is more frequent than paralysis.

The manner in which lead is handled makes a vast difference so far as the liability to plumbism is concerned. Thus Stüler found in Vienna that carriage painters are ten to twenty times more subject to lead poisoning than house painters. This has been confirmed by Edsall in this country. The reason for this is that carriage painters apply a large number of coats of paint and varnish, polishing between each coat, and thereby enveloping themselves in dust which contains much lead; furthermore, carriage painters are required to work indoors.

Red Lead (Litharge, Massicot, or Lead Oxid).—In the manufacture of red lead the metal is simply roasted in a reverberatory furnace and raked from time to time. A considerable amount of fumes escape from the mouth of the furnace and unless this is hooded and a strong draft provided to carry it away the workmen may become poisoned. The red lead is removed in large pieces and then ground, during which process quantities of fine dust are raised which may also poison the workmen.

White Lead.—Most of the white lead is still made by the old Dutch method, which consists in the transformation of metallic lead into the white carbonate by a slow and double process of conversion. Numerous earthenware pots containing 3 per cent. of acetic acid are placed on tan in a large three-walled chamber, and upon these pots are laid thin strips of metallic lead and subsequently planks of wood. Tier after tier of pots resting on bark and covered with metallic lead and wood are thus superimposed until the chamber, 25 or 30 feet in height, is filled to within 6 feet from the top. This chamber, known as the "blue" bed, is

FIG. 135.—A WORKER WITH LEAD OXID, SHOWING RESPIRATOR TO PROTECT HIMSELF AGAINST THE POISONOUS DUST. (Mass. State Board of Health.)

kept closed for 14 weeks or longer. Fermentation causes a rise in temperature and a production of carbonic acid. The acetic acid acts upon the lead and converts it into acetate of lead, while the CO_2 evolved from the bark changes the acetate into carbonate or the well-known white lead of commerce. The danger of plumbism occurs during the emptying or stripping of what is now called the "white" bed. If sufficient time has not been given for the very soluble acetate to have become changed into the carbonate the danger is thereby greater. During the stripping of the "white" bed there is a considerable quantity of dust raised, a large part of which is white lead, and unless spraying with water is effectively carried out the workmen cannot avoid inhaling the dust.

Dr. T. Morison Legge found that of 1,463 persons employed off and on in white lead works the incidence of lead poisoning was 6 per cent. of the average number regularly employed, and in those casually employed 39 per cent. This shows the great risk of exposing unskilled labor in a dangerous occupation.

In some progressive plants the white lead is transferred mechanically from the white beds to the mixing department, where it is ground, washed with water, and subsequently mixed with oil, and thus converted straightway into paint without even being handled at all. This greatly diminishes the danger.

White paint contains 75 per cent. of lead carbonate and 25 per cent. of oil. The men who mix the paint do not suffer to any extent from plumbism, as little dust is raised during this process.

The Manufacture of Pottery and Earthenware.—Next to the white and red lead industries the glazing of pottery and earthenware furnishes the largest numbers of victims of lead poisoning. The lead is contained in the glaze with which such ware is coated and the danger occurs in cleaning and polishing the "biscuit," during which process a considerable amount of dust containing lead is raised.

The article to be made is shaped and molded from the clay or kaolin and then placed in an oven and fired. Some pottery, such as terra cotta and stoneware, requires only one firing, but all others have to be fired twice. After the first firing the ware is known as "biscuit." The biscuit is dipped into a liquid glaze and then fired again, which produces the hard, smooth, vitrified surface.

A "non-fritted" glaze contains raw lead, that is, the carbonate. The ware is dipped into the mixture, then dried, and each piece is smoothed and cleaned. During the cleaning of the biscuit, especially when prepared with a non-fritted glaze, considerable dust containing lead carbonate is raised.

A "fritted glaze" is a compound of raw lead (carbonate), silica, boric acid, etc., fused together at a high temperature. This produces a glass-like substance in which the lead is rendered more insoluble. When ground and mixed with fine clay and water it forms a white chalky liquid into which the biscuit is dipped. This fritted glaze is safer for the workmen than the non-fritted glaze containing raw lead.

The use of leadless glazes has been opposed by many manufacturers, but it has been shown by Thorpe and Oliver that the largest proportion of earthenware can be made without lead in the glaze. The advantages of lead in the glaze are that it melts at a low temperature and gives a deep gloss with a delicate bluish tint which is generally admired. In justice to the manufacturers it should be stated that they have not found the leadless glazes to prove satisfactory.

In the manufacture of pottery the workmen are liable to lung dis-

eases on one hand, and lead poisoning on the other. This places pottery manufacture high on the list of dangerous trades. "Potter's rot" and "potter's asthma" are familiar terms. The dangers may largely be avoided by the introduction of fans and strong drafts to carry away the dust from the faces of the workers and the use of a fritted or, better, a leadless glaze.

Besides the glazing, exposure to lead occurs in the decorating of pottery, in putting on dry colors, and in so-called erographing, in which the lead colors are sprayed on the surface of the pottery.

Another danger in the manufacture of pottery is the irritating dust which rises from the finely ground flint in which the biscuit is packed when fired.

File Cutting.—The better grades of files are cut by hand in the following manner: The workman sits astride on a "stock." In front of him is a stone block, in the center of which a piece of steel bar called a "stiddy" is inserted, and in this stiddy is placed a piece of metallic lead which is called the "bed." The lines are made by striking with a hammer and chisel, each line upon the file representing a blow from the hammer. There is a considerable quantity of dust given off when the file is rubbed with charcoal before it is turned. This dust contains a large proportion of lead. The lead is also taken into the mouth from the hands through uncleanly habits of the workmen. File cutters frequently suffer from lead palsy.

Miscellaneous Industries.—Layet computed that in France 111 industrial processes involve the use of lead. Hamilton[1] found 70 such processes in Illinois in which lead or its salts are handled and which have caused lead poisoning in recent times.

Some of the industries in which lead poisoning may occur are: making and selling wall paper, polishing brass, polishing nickel, finishing cut glass, holding lead-covered nails in the mouth while shingling a roof, working with aluminium foil (7 per cent. lead), in lithography, zinc smelting, making ornamental tiles with a non-fritted lead glaze, wrapping cigars in "tin" foil, enameling bath-tubs, laying electric cables, stopping the inequalities of wood with white lead in making automobiles, assembling and recharging old storage-battery plates, polishing handles of coffins, etc. Lead poisoning may also be contracted from diamond cutting, the setting and polishing of precious stones, from enameling iron plates and hollow ware, from electric accumulator works, from printing, type-founding, type-setting, and linotyping, from dye works where yellow colors are got from chromate of lead, from house, coach and ship painting, etc.

[1] *J. A. M. A.*, Vol. LVI, No. 17, April 29, 1911, 1240-1245.

PREVENTION

The prevention of lead poisoning rests, in the main, upon the fact that the lead comes to the workman usually as dust, sometimes as fumes through the air, but it must be remembered that lead is also carried to the mouth by deposits on the hands and other objects. Whether the lead enters the body by inhalation, by ingestion, or through the skin becomes more of an academic than a practical question so far as prevention is concerned.

The first essential then is to keep the air which the workman breathes and which surrounds him free of lead. Most cases of lead poisoning could be averted by a proper system of ventilation. Certain processes should be carried on under hoods with a strong draft, or in cabinets, or special rooms with an air current so arranged that the lead is kept away from the mouth, nose, hands, and clothes of those who are exposed.

On the part of the workman the prevention of lead poisoning consists in cleanliness of the hands and of the finger nails, frequent bathing, and the use of special clothing while at work. Care must be taken not to carry the fingers, which may be contaminated with lead, to the mouth and nose, and to thoroughly wash the hands before eating. Workmen should never take their lunch in the rooms where there is a suspicion of lead in the air. In the few instances where the above precautions are not practical respirators should be worn.

Cleanliness is one of the all-essential requirements. A special room for the clothes of the workmen and special overalls should be provided for those who are exposed to lead. It is ignorance of the danger and the want of personal cleanliness that make casual labor in lead works especially dangerous. Even the women who wash the clothes of the workmen employed in lead factories may sometimes suffer from lead poisoning. Lavatories should be provided at the factory and the hands should be washed with water containing a small quantity of acetic acid followed by a liberal allowance of soap.

Workmen should alternate employment and not remain too long in the dangerous departments. Supplanting hand labor by machinery diminishes the number exposed to the risk. A medical inspection is an important preventive guard in educating the workmen and in detecting mild and beginning cases.

A radical measure would be the substitution of zinc-white for lead paints. Zinc may be used as a substitute for lead, especially in indoor work; in fact this has been required by law in France. White lead appears to be superior to zinc for outdoor work.

Keeping down the hyperacidity of the gastric juice is believed to be a good preventive measure. This may be accomplished in part by taking a bland oil or drinking milk rich in cream at intervals during work.

The Massachusetts State Board of Health issues the following protective measures against lead poisoning:

The poison gains entrance into the system:

(1) By swallowing minute particles of lead.

(2) By inhaling lead dust or the fumes of lead in a molten state, or the vapor of lead in a fused state.

(3) By absorption from the skin in handling lead.

FIG. 136.—THE STONE INDUSTRY. The workman is using a surfacing machine operated with compressed air. The strong blast of air keeps the granite clean, but gives rise to a great amount of dust. Of the mineral dusts granite is generally considered as most irritating. (Mass. State Board of Health.)

Advice to Employees

(1) General personal cleanliness is of the first importance.

(2) Thoroughly clean your hands before touching food and before leaving the workroom.

(3) Thoroughly rinse your mouth before eating.

(4) Take good, nutritious food and plenty of milk.

(5) Take a substantial breakfast; an empty stomach is more susceptible to the poisonous effects of lead.

(6) Never eat at your work. Eat your luncheon outside of the workroom if possible; if not, in a part of the room away from the lead. Never smoke or use tobacco in any form while at work.

(7) Avoid all excesses; alcoholic beverages are especially injurious.

(8) Wear overalls or a long coat at your work; also a cap or some head covering. Whenever practicable wear gloves when lead is to be handled.

(9) Persons working in white lead or other powdered compounds of lead should always wear a respirator while at work. Cause as little dust as possible.

(10) Consult a physician at the first sign of ill health.

Advice to Employers

(1) Provide washing facilities, lockers, and a place for the employees to eat luncheons away from lead.

(2) Provide respirators for all the workers who have to handle white lead or other powdered compounds of lead.

(3) The floors of the workrooms and benches at which men work should be cleaned daily after thoroughly moistening them.

(4) These regulations should be posted in a conspicuous place in the workroom.

PHOSPHORUS

There are two kinds of phosphorus: (1) the white or yellow, discovered by Brandt of Hamburg in 1669, (2) the red or amorphous, discovered by Schröter of Vienna in 1845. The amorphous phosphorus is obtained from the white phosphorus by exposing it in a closed vessel for some time to a temperature of 250° C. The white or yellow phosphorus is poisonous and has been the cause of much suffering in the match industry. The red or amorphous phosphorus is practically not poisonous.

Three kinds of matches are made: (1) the safety match, which contains no phosphorus and is harmless. The match heads contain potassium chlorate or chromate and other compounds rich in oxygen from which the oxygen required to induce conflagration is evolved. The paste applied to the side of the match-box contains antimony sulphid and red phosphorus: (2) the strike-anywhere match contains the poisonous white phosphorus in the head, and in addition glue, chlorate of potassium, powdered glass, and magenta or some other coloring agent. The paste, or composition, contains on an average 5 per cent. of phosphorus. It is in mixing this paste, especially when done by hand in open vessels, and also in dipping the wooden splints, that the work-people are exposed to fumes that become a menace to health: (3) the strike-anywhere match made with the non-poisonous sesquisulphid of phosphorus. The paste from which these non-poisonous matches are made is as follows:

Sesquisulphid of phosphorus	6 parts
Chlorate of potassium	24 "
Oxid of zinc	6 "
Red ochre	6 "
Powdered glass	6 "
Glue	18 "
Water	34 "

Sometimes these matches also contain from 3 to 4 per cent. of red phosphorus which prevents the formation of unstable subsulphids. These matches have been used exclusively for the last 12 years in France. In our country the strike-anywhere match made with poisonous white phosphorus constitutes the principal output.

When pure, phosphorus is colorless and transparent, but when exposed to the light it becomes yellowish. The white and yellow forms are extremely poisonous; the red or amorphous phosphorus can be handled with impunity. Red phosphorus does not take fire when rubbed on a rough surface. It is non-volatile and when swallowed is, comparatively speaking, non-poisonous. One to 3 grains of white phosphorus will cause death. The fumes from white or yellow phosphorus are rich in phosphorus oxids and these are absorbed in various ways. Professor Thorpe exposed decayed human teeth to the fumes of phosphorus for 12 hours and he found that they lost 0.37 per cent. of their weight and that carious teeth, when exposed to a dilute solution of phosphoric acid (1 per cent.), lost 8.9 per cent. of their original weight. The atmosphere of an ill-ventilated match factory in which the white or yellow phosphorus is used reeks with the garlicky odor characteristic of phosphorus. The fumes become dissolved in the saliva of the mouth and exercise a solvent action upon the teeth of persons inhaling this poisonous atmosphere several hours of each working day. The poison also clings to the fingers and hands of the work-people. Thus Oliver found the hands of a boxer to be deeply stained by the dye given off by the heads of the matches, to emit a characteristic garlicky odor and glow in the dark. The phosphorus is also absorbed by the drinking-water if exposed.

The principal and characteristic disease produced by white or yellow phosphorus is necrosis of the jaw, known as "phossy jaw." This is a localized inflammatory infection of the jaw-bone extremely painful in the early stages, which runs a chronic course and invariably ends in the localized death of the bone. The gums become swollen and the jaw-bone painful; sooner or later pus forms and although the teeth are extracted the pain continues. The inflammation gradually extends to the bone.

It is probable that the phosphorus fumes and the phosphoric acid acting through decayed teeth set up inflammation, thereby allowing microorganisms, always present in the mouth, to carry the morbid process

deeper. Lewin of Berlin does not believe that it is primarily necessary for a lucifer match-maker to have decayed teeth, for the phosphorus fumes, in his opinion, inflame the gums in the first instance, and as a consequence there is induced a septic gingivitis, which is followed by disease of the bone. Other effects of phosphorus, though less frequent, are phosphorism, which is a general cachectic condition met with principally in female workers, and characterized by anemia, dyspepsia, albuminuria, and a tendency to bronchitis. *Fragilitas ossium* is another condition met with in phosphorus workers. According to Dearden, the bones of match-dippers contain an excess of phosphoric acid which combines with the preëxisting neutral phosphate of lime to form a slightly acid salt and thereby causing excessive brittleness of the bones.

The prevalence of phossy jaw may be gleaned from the fact that in Great Britain somewhat less than 1 per cent. of the match-makers have suffered from it. In Switzerland the incidence was formerly 1.6 to 3 per cent., and in France 2 to 3 per cent. In this country the subject was investigated by the Bureau of Labor, who found that there are about 3,500 employees in 15 of the 17 match factories in the United States; of 3,383 whose occupation was specified 65 per cent. were exposed to phosphorus fumes. It was also found that 95 per cent. of 1,395 so exposed are women. An intensive study of three factories was made and 82 cases of necrosis were discovered.

Several years ago the Belgian government offered a prize of 50,000 francs to any person who would invent a safety strike-anywhere match free from white phosphorus. The problem was solved by Sevène and Cahan of France, who demonstrated that the sesquisulphid of phosphorus would accomplish all that white phosphorus does without causing poisoning. The sesquisulphid is an almost inodorous powder and is, practically speaking, nonpoisonous. It contains a trace of red or amorphous phosphorus. Since the introduction into France of the manufacture of the sesquisulphid match there has not been in the factories of that country, where the manufacture of matches is a state monopoly, one case of phosphorus poisoning, nor has there been any explosion or fire in any of the match works. It has been found that the sesquisulphid of phosphorus acts, in some instances, as an irritant, causing conjunctivitis and edema of the eyelids, also eczema of the skin. This may be obviated by bathing the eyes and douching the nostrils twice a day before leaving the factory with an alkaline solution of bicarbonate of soda.

PREVENTION

The prevention of phosphorus necrosis consists in the substitution of the sesquisulphid for the poisonous white and yellow forms. Even stringent regulations will not protect those who have to work with the

white or yellow phosphorus. The experience of Great Britain is especially noteworthy. In 1888 Great Britain, after a thorough investigation of the conditions, made stringent regulations coupled with an efficient inspection, but phossy jaw was not prevented, and in 1908 the use of white phosphorus was prohibited. White phosphorus was prohibited by Finland in 1872 and in Denmark 2 years later, since which time no case of phosphorus necrosis has occurred in these countries. France prohibited its use in 1897, Switzerland in 1898, the Netherlands in 1901, and in 1905 the International Association for Labor Legislation secured a

FIG. 137.—WORKMEN EXPOSED TO ZINC FUMES IN BRASS CASTING, CAUSING A CONDITION KNOWN AS "BRASS-FOUNDER'S AGUE."

treaty providing for the prohibition of the making and selling of matches made of white phosphorus, which treaty was signed by France, Denmark, Germany, Italy, Switzerland, the Netherlands, and Great Britain. This country has grossly neglected to protect its workmen and has yet taken no action despite the serious conditions demonstrated by John B. Andrews, Secretary of the American Association for Labor Legislation.[1]

Phosphorus poisoning may in part be prevented by a medical examination, special attention being paid to the state of the teeth. The services of a dentist are essential. Personal cleanliness and the use of mouth washes are helpful. The workroom should be well ventilated and

[1] Congress passed a prohibitive tax on poisonous phosphorus, April, 1912; effective June, 1913.

fans should be kept running to withdraw all fumes away from the faces of the workers. Washing accommodations should be ample, hot and cold water should be provided, along with plenty of soap and towels.

Persons sometimes commit suicide by dissolving the white or yellow phosphorus from match heads and drinking the solution. This is not possible in the case of matches made with the red or amorphous phosphorus or the sesquisulphid.

ARSENIC

Arsenic acts as an irritant to the skin and mucous membranes, setting up conjunctivitis, eczema, and ulcerations; it also produces general poisoning, causing anemia and neuritis. Arsenical neuritis is particularly severe and often serious. In the industries arsenical poisoning is found among workers in the manufacture of Scheele's green, in the manufacture and use of wall papers and artificial flowers containing arsenical coloring agents, during the packing of white arsenic, and in reduction works of arsenic mines.

Workers in arsenic suffer from painful redness of the eyes and from eczema of the eyelids. Men employed in the manufacture of Scheele's green (arsenite of copper) frequently have painful ulcers on their fingers or other portions of the body where the dust collects. Smelters sometimes suffer from "arsenic pock," an irritation of the skin due to the action of the very fine dust upon the perspiring skin. The bronchitis from which workers about smelting works suffer has been attributed partly to the fumes given off by the raw material and partly to the rather large amounts of sulphur contained in the fumes. Men employed in removing vitriol solution from the depositing tanks in copper works occasionally suffer in consequence of the inhalation of arsenuretted hydrogen gas.

Arsenic is also used in curing furs. The Massachusetts law forbids arsenic to exceed 1 grain per square yard, but analyses reveal that it often reaches 170 grains. Out of 42 samples of fur recently examined in America 11 were found heavily loaded with arsenic. The presence of such large quantities of arsenic in furs that are worn or in rugs for rooms must be a source of danger.

From wall paper the arsenic may be absorbed either as solid particles detached from the paper or as a volatile gas formed from arsenical organic matter by the action of several moulds, notably *Penicillium brevicaule, Mucor mecedo,* etc. (Gosio.) For the liberation of the volatile arsenical compounds moisture and a certain amount of heat (60 to 95° F.) are necessary. The volatile compound, according to Sanger, is probably an organic derivative of arsenic pentoxid. Arsenic, as well as other irritants, is believed to predispose the tissues to growths of a cancerous nature.

MERCURY

Mercurial poisoning may be contracted by workmen employed in extracting mercury from cinnabar (sulphid of mercury), in which it is usually found in nature. The ore is simply roasted and the mercury volatilizes and readily condenses in metallic form. Mercury volatilizes at a low temperature and it is this circumstance which creates much of the danger to those who work with this substance, especially men who work in a closed and heated atmosphere containing the vapor given off by the metal. Mercury is absorbed by the digestive system, the respiratory tract, and also through the skin. As an instance of the absorption of mercury through the skin Edsall cites two cases in dentists who were poisoned as a result of the custom found in many dentists of working up their amalgam in the palms of their hands.

The occupations in which mercury is used and in which mercurial poisoning occurs are: the separation of gold and silver from their respective ores, which is done by means of an amalgam; the manufacture of incandescent lamps, in which mercury pumps are used to create a vacuum; in barometer and thermometer making; in felt-hat and fur dressing, in which mercuric nitrate is used; in water-gilding, where an amalgam of gold or silver, after having been applied to an object, is heated and the mercury driven off; and other industries.

The New York and New Jersey section of the National Civic Federation in three months' time found 60 cases of mercurial poisoning, a nervous disease called in the trade "the shakes," among the hat makers of Brooklyn, Newark, and Orange as a result of the mercury salts used in preparing felt.

The symptoms of mercurial poisoning are: anemia, headache, dizziness, tremor of the muscles, especially the tongue and limbs, fetid breath, soft, swollen, and ulcerated gums, and loosening of the teeth. The submaxillary and other glands of the neck become painful and the secretion of saliva excessive. Erethism and apprehensiveness are common; in severe cases depression and melancholia. A persistent and apparently causeless diarrhea is frequently a symptom of mercurial poisoning.

PREVENTION

The prevention of mercury poisoning is almost a direct counterpart of the prevention of lead poisoning. The air must be kept free of mercury, and this can be accomplished by proper systems of ventilation, by the use of hoods with forced draft and other devices to keep the mercury fumes away from the workmen. Rubber gloves may be worn to prevent absorption through the skin and also to prevent the carrying of the mercury to the mouth. Here again scrupulous cleanliness in and after leav-

ing the workroom, a change of clothing, and washing the hands before eating are essential.

CARBON MONOXID

Carbon monoxid is a colorless, inodorous, and highly poisonous gas. It burns with a pale blue flame. It is one of the products of the incomplete combustion of illuminating gas, also of coal and explosives. It is met with in coal mines and other subterranean galleries where blasting has been effected by dynamite and gun-powder. It forms 7 to 10 per cent. of ordinary illuminating gas (coal gas) and 30 per cent. of water gas. It is the source of the blue flame seen on the surface of an ordinary coal fire. The gas is given off in quantities from coke ovens; it is evolved from blasting furnaces in the smelting of iron, especially during the charging of furnaces and their tapping. Carbon monoxid frequently remains in the furnace, and workmen who enter such a furnace in order to clean it may be overcome. In England the law requires two workmen to clean furnaces; one stands by in case of accident. Carbon monoxid is also evolved from hot-water heaters; in the Leblanc process of soda manufacture; in cement and brick works, etc.

The poisonous properties of carbon monoxid are, according to Haldane, due to the great affinity it has for the hemoglobin of the red corpuscles. It has from 140 to 250 times greater chemical affinity for hemoglobin than oxygen. It forms carbon monoxid hemoglobin, a more stable compound than oxyhemoglobin, and therefore prevents the oxygen being given to the tissues. When the percentage of carbon monoxid rises to 0.4 the atmosphere becomes dangerous to animal life. (See page 635.)

The inhalation of carbon monoxid causes headache and a sense of loss of power in the lower extremities. It is this circumstance which explains many of the cases of fatal poisoning in confined spaces. There are also dizziness, throbbing of the temples, ringing in the ears, a sense of lassitude, and, in severe cases, convulsions and loss of consciousness. The inhalation of small quantities also leads to delusions and other mental symptoms. If the gas enters a bedroom and is inhaled by persons who are asleep the sleep only becomes deeper and profound narcosis is developed from which there may be no awakening.

Oliver gives the following illustration of the subtle poisoning by carbon monoxid at Pelton Fell, a mining village in Durham County. Some shale which had been tipped at the edge of a ravine caught fire. The carbon monoxid gas given off during the combustion traveled through the soil and entered two houses in different streets, full 30 feet away, causing the death of two elderly people. It is to the breathing of this gas during sleep that the death of tramps, drawn to the coke ovens by their inviting warmth on a winter's night, is attributed. I

have already instanced the case of death from carbon monoxid resulting from the imperfect operation of a gas water-heater. (See page 636.)

HYDROGEN SULPHID

Hydrogen sulphid is an extremely poisonous gas causing death instantaneously if inhaled in large quantities. In smaller amounts the symptoms caused are nausea, vertigo, headache, general malaise, all of which soon disappear if the workman goes into the open air. There are only a few industrial undertakings in which hydrogen sulphid is met with, such as chemical and gas works; the black bronzing of metals by means of sulphid of arsenic; the cleaning of boilers; in certain processes of soap making where large quantities of fat are decomposed; in the preparation of Prussian blue; during the decomposition of ferrocyanid of potassium by sulphate of iron. In nature hydrogen sulphid is one of the products formed during the putrefaction of organic matter containing sulphur. The gas may therefore be found about privies, the mud of marshes, and collections of filth and manure, but in quantities too small to seriously influence health.

DUSTY TRADES

Dust is the great enemy of the workman. Much ill health is caused by the inhalation of dust, some of which is also injurious when ingested and some of which is irritating to the skin. Dust of all kinds, both organic and inorganic, is met with in the various industries. Organic dust is usually less irritating and dangerous than inorganic dust, which becomes harmful particularly when the particles are sharp and therefore irritating. The principal trades and occupations in which excessive amounts of dust are found are: all forms of grinding and many processes of polishing and cleaning; the textile industries; in the lead, copper, and iron trades irritating and poisonous dusts are raised; also in pottery works and masonry, and in the handling of leather, skins, feathers, wool, cotton, wood, paper, tobacco, etc. The amount of dust may be very great; thus Hesse found in one cubic meter of air the following amounts of dust in the occupations named:

Felt hat factory	175	milligrams
An old flour mill	48	"
A new flour mill	4	"
Mechanical knitting	3	"
Sculpturing	9	"
A paper factory	4-25	"
Iron works	72-100	"
A coal mine	14	"
A living room	0	"

The kinds of dust vary greatly in their hygienic significance. Some are poisonous, some act as mechanical irritants. The principal poisonous dusts found in the industries are lead, mercury, arsenic, phosphorus, and zinc; less often substances from tobacco, wood, dyes, and chemical works. The dust particles which act by mechanical irritation are especially the hard, irregular particles with sharp edges from iron, steel, and other metals; from granite, basalt, or marble; while those from coal, chalk, and plaster of paris are less irritating.

According to Sommerfeld the following proportion of persons per

Fig. 138.—A Very Dusty Trade—Drum with Nails Which Combs out the Small Pieces of Broom Corn. (Mass. State Board of Health.)

thousand in the various dusty occupations mentioned die of pulmonary tuberculosis:

Occupation without dust production	2.39
With dust production	5.42
With porcelain dust	14.0
With iron dust	5.55
With lead dust	7.79
With stone dust	34.9
With porcelain dust	14
With stone workers	4.3
With wood and paper dust	5.96
With tobacco dust	8.47

Persons exposed to excessive amounts of dust for long periods of time suffer from a general condition known as *pneumonokoniosis;* when due to coal dust the condition is known as *anthracosis;* when due to stone dust, *siderosis* or *chalicosis;* when due to vegetable fibers such as cotton, *byssinosis.* The dust may, in part, be free in the alveoli of the lungs and in part is inclosed in the cells. The epithelial cells lining the alveoli act as phagocytes. At times some of the alveoli may be plugged with dust particles. Sometimes the dust remains in the lungs without any apparent reaction on the part of the tissues. Usually round cells appear in the interalveolar spaces, and other indications of irritation and inflammatory reaction take place, leading to connective tissue formation between the alveoli and thickening of the alveolar wall itself. This may progress to an indurative bronchitis; that is, several alveoli become drawn together by the contracting connective tissue into a nodule. Other forms of inflammation, such as nodular peribronchitis or nodular perivasculitis, may take place. The dust particles are also carried by the phagocytes to the regional lymphatics, where they lodge. These irritative processes cause a low grade inflammatory reaction which only awaits the coming of bacteria to start specific or destructive processes.

Some dust is especially irritating to the conjunctiva, as wood dust or arsenic. Certain kinds of dust are prone to cause chronic catarrhal inflammation of the upper respiratory passages, while dust containing specific microorganisms such as anthrax may lead to acute pneumonia (wool sorter's pneumonia).

General Principles of Prevention.—Much of the dust raised in industrial processes may be limited by improvements in machinery or preventive devices. Sometimes the dust may be kept down by moisture, sprays, or even conducting the work under water when practicable. Thus wet grinding may be substituted for dry. Certain dusty operations should be conducted in inclosed hoods or special cabinets so as to confine the dust and thus protect the workpeople, or the dust may be removed by suction fan devices. Good ventilation diminishes the danger very much. When workmen are compelled to stay in dusty atmospheres they should wear respiratory masks, and the number of persons thus exposed should be reduced to a minimum. Some exceedingly dusty processes, such as cleaning castings with a sand blast, demand the wearing of a protective headgear. Many workmen prefer taking chances to wearing uncomfortable respirators.

THE TEXTILE INDUSTRIES

The manufacture of cotton, linens, silk, and jute has received an unenviable reputation as dangerous occupations, despite the fact that these industries need not in themselves be particularly unhealthy occupa-

tions. The textile industries illustrate several points in the diseases of occupation. One is that an entire industry should not be condemned because one of its processes is attended with a certain amount of danger. The other is that the risks to health may be prevented or greatly ameliorated. General improvement in the sanitary conditions of textile mills is one of the promising signs of material advancement in industrial hygiene.

The principal conditions which affect health in the textile industries are: The working in a dusty atmosphere which is often kept very moist and usually very warm in order to keep the fiber pliable and workable. The humidity and temperature may be regulated, and by efficient systems of ventilation their ill effects may be minimized or even neutralized. The dust may also be lessened, and in the processes in which it is excessive the workmen may protect themselves with respirators.

Much dust is raised during the opening and emptying of the bales of the raw material. This is avoided in the better mills by the use of machinery. Most of the dust is raised during the process of "carding"; some during "roving," "spinning" the yarn, and "winding" it; and also considerable during "weaving." In linen factories the "hecklers," that is, the men who dress and sort the rough flax (converted into tow by having been passed through a machine), are exposed to considerable amounts of dust and suffer from dryness of the throat and bronchitis, attended by cough and shortness of breath. In the manufacture of sacks, twine, and carpets from jute the processes that are extremely dusty are the preparing and spinning. The dust given off by jute is irritating.

Working in an atmosphere which is excessively moist and frequently very warm, and, further, containing an excessive amount of organic dust, subjects the workmen to artificial and unnatural conditions which cannot be conducive to health. Presumably the heat and moisture predispose to rheumatic states and inflammatory conditions of the respiratory tract which are aggravated by the irritation of the fibrous dust. It is believed that workmen so exposed are more prone to contract common colds, bronchitis, tuberculosis, and other inflammatory diseases of the respiratory tract.

In Massachusetts there is a law regulating the amount of humidity and temperature in the textile mills which is based upon the English schedule contained in the Weaver's act of 1870. The conditions in Massachusetts, however, are so different from those found in England, especially in the summer time, that the schedule has not been found practical. Much of the ill effects in the textile industries may be neutralized by good ventilation, abundant air space, cleanliness, sufficient light, and the use of improved machinery. Special rooms should be provided for the clothes, in order that the moist garments may be

changed for dry ones before the workpeople go into the open air, thus avoiding the chilling effects of damp garments.

WOOD DUST

It is well known that workers in wood are subject to the mechanical effects of ordinary sawdust, which is moderately irritating. Workers with boxwood, teak, and sequoia (redwood) are subject also to the general poisonous effects of alkaloids and other substances contained in these woods which may have more marked general effects, especially on the circulation and, still more frequently, marked local effect on the mucous membranes and the skin. In 1902 Young observed that men working with Maracaibo boxwood complained of dryness of the throat and inflammation of the eyes which lasted two or three days. This wood is used in the making of rulers. Oliver notes that joiners that saw and chip sequoia wood suffer with symptoms resembling a bad cold in the head and chest; a tolerance seems to be established except by men who are liable to bronchitis and asthma. Wounds caused by splinters of the wood invariably suppurate and do not heal readily. Oliver found that rats were also susceptible to sequoia sawdust. They suffer from a running at the nostrils.

Certain kinds of wood have a bad reputation among joiners. Some sawdusts are more irritating than others, probably from the large amount of inorganic matter they contain. A West African boxwood from which shuttles are made causes headache, coryza, excessive secretion of tears, and attacks of asthma. These woods contain alkaloids, glucosides, and other extractives. Workers in teakwood occasionally suffer from dermatitis.

MINING

Coal mining is one of the dangerous and unhealthy occupations. The dust, the unnatural conditions under which the miner is compelled to work underground, the poor air, and sometimes exposure to poisonous fumes all conspire to make this occupation one attended with unusual risks. The unsatisfactory methods for disposal of feces often found in mines favor the spread of hookworm and other parasites. To this must be added the danger of accidents and explosions.

The conditions of mines have been greatly improved, especially through better systems of ventilation, through the use of safety lamps, through reduction of the amount of dust, the regulation of the hours of occupation, and devices to detect poisonous and explosive gases. The sanitation and cleanliness of mines have also shown development. As an illustration of some of the complications and difficulties of this subject, reference is made to the fact that moisture will prevent explosion

in mines. Moisture was, therefore, introduced into some of the German mines with good results, so far as explosions are concerned, but the moisture favored the development of the hookworm larvæ and hence caused such a great increase in the amount of hookworm infection that it became necessary to seek other methods.

The mortality from accidents and diseases of the lungs is high. Coal miners' phthisis, or anthracosis, is a well-known disease. Although coal is a vegetable product the result largely of microbial action, fresh coal is free from microorganisms. Oliver points out that in some of the mining centers colliers not only suffer less from pulmonary tuberculosis than persons in other occupations, but that they also suffer unequally in different mining centers. Why this is so it is difficult to say. While the death rate from pulmonary tuberculosis in miners is in some places low, that due to non-tuberculous affections of the lungs is, comparatively speaking, high.

DeCrocq speaks of the rarity of phthisis among Belgian coal miners. Arnold reports that in Germany tuberculous diseases are rare among coal miners and that there is a prevailing opinion that anthracosis is antagonistic to tuberculosis. Goldman attributed the freedom of the coal miner from pulmonary tuberculosis to an antiseptic action of the coal dust.

Other diseases to which coal miners are subject are "beat hand," as a consequence of using the pick and friction of the handle. The skin of the palm over the bases of the fingers of both hands, also the skin over the fleshy ball of the thumb and that of the other side of the hand, becomes extremely hard and horny. In addition to the enormous thickening of the epithelial layers of the skin there is inflammation of the subcutaneous connective tissue. Occasionally suppuration takes place in the deeper layers of the hard skin. The suppuratory areas are called "keens" by the miners. Beat hand is a painful affection and unfits the individual for work for some time. A similar condition sometimes occurs on the knees and elbows, hence the term "beat-knee" and "beat-elbow." Miners also frequently complain of backache, largely the result of the peculiar mode of sitting while at work. Dyspepsia, miner's nystagmus, and ankylostomiasis are other conditions to which miners are prone.

EFFECTS OF HEAT

In many trades workmen, more particularly firemen, stokers, workers in foundries and steel mills, are exposed to high degrees of heat. Edsall[1] has recently called attention to the ill effects of exposure to unusual degrees of heat. The symptoms are acute and violent muscle spasms.

[1] *Jour. Amer. Med. Assn.*, LI, Dec. 5, 1908, p. 1969.

The acute effect may be heat-stroke and heat prostration; there may be nervous lesions such as focal meningitis, as well as more or less serious circulatory weakness, anemia, acute and chronic disturbances of digestion, acute and chronic nephritis. Respiratory diseases and skin lesions appear to be unduly frequent in persons exposed to high degrees of heat. There is more than a suspicion that cataracts, retinal and choroidal changes, or chronic conjunctival lesions are brought on in glass-blowers and perhaps also in iron puddlers and other persons whose eyes are exposed to very intense heat and light. De Schweinitz states that he can often tell whether men working at puddling furnaces are right-handed or left-handed by studying the effects of this exposure on their eye grounds. Ropke[1] states that Quint described to him cases of right-sided cataract in right-handed iron workers and left-sided in those who were left-handed.

PARASITES

There are several species of parasites to which workmen in certain industries are specially subjected. Of these the best known are: anthrax, or wool-sorter's disease, and hookworm disease, or miner's anemia.

Wool-sorter's disease is an infection with the *Bacillus anthracis*. The spores cling to the hides of animals that have died from the disease or have been slaughtered on account of it. Spores also remain attached to wool and horsehair and to pig's bristles used in brush-making. The infection may be taken in through the slightest scratch or any open wound or through inhalation of dust containing the spores, or may be ingested in the food. Wool-sorter's disease most often appears in the wool-sorting, wool-combing, and spinning industries, in the manipulation of horsehair for stuffing chairs and mattresses, and the preparation of bristles for brush-making. Anthrax has also been met with in persons employed in tanyards and in warehouses that connect with docks. The subject is fully discussed by Legge in his Milroy lectures.[2]

The prevention of anthrax is first and foremost a problem in animal husbandry which, in this country, comes under the purview of the Bureau of Animal Industry. Animals having anthrax should be killed and all anthrax carcasses should be buried, incinerated, or tanked in such a manner as to destroy the infection and prevent its dissemination. This is one of the questions for international sanitary agreement, for the wool from Prussia, the hair and mohair from Asiatic Turkey, the horsehair from China, the bristles from Siberia, and the hides from India may carry the anthrax spores from these far-off lands and cause infection among our workmen. It is exceedingly difficult to disinfect

[1] Weyl's "Handbuch der Arbeiterkrankheiten," 1908.
[2] *Lancet*, March 18, 1905.

hides so as to kill the anthrax spores. Horsehair, cowhair, goat's hair, pig's bristles, and wool before they are manipulated should be disinfected either: (1) by steam at 17 pounds pressure, equivalent to 220° F., for at least half an hour; (2) by boiling for at least a quarter of an hour in a 2 per cent. solution of potassium permanganate, and subsequent bleaching in a 3 to 4 per cent. solution of sulphurous acid; (3) by boiling in water for at least two hours. In Germany the government regulations require the disinfection of wool and hair from foreign parts and provide public disinfection stations for this purpose. For a discussion of anthrax see page 285.

Hookworm Disease.—Miners are specially subject to hookworm disease. The parasite enters through the skin from the polluted soil of the mines. The outbreak which called attention to this danger was the epidemic which occurred among the workmen on St. Gothard's tunnel in 1892. Since then the disease has been called "miner's anemia." Gunn[1] found that from 50 to 80 per cent. of those working in the mines of California and the neighboring state of Nevada were infected with hookworms. For a full discussion of hookworm disease see page 114.

Other occupations in which there is a special exposure to the risk of infection are: physicians, nurses, ward-tenders, pathologists, experimental investigators, etc.

THE CAISSON DISEASE

The effects of compressed air and the effects of rarefied air are discussed on pages 598 and 600.

REFERENCES

Oliver, Thomas: "Diseases of Occupation, from the Legislative, Social, and Medical Points of View." New York, 1909.

Weyl, Theodor: "Handbuch der Hygiene." Gewerbehyg., Vol. VIII, Jena, 1897.

Various authors: "Risks in Modern Industry." Published by the American Academy of Political and Social Science, Phila., Pa., 1912.

Goldmark, Josephine, and Brandeis, L. D.: "Fatigue and Efficiency." Charities Publication Committee, 105 E. 22d St., New York, 1912.

[1] *J. A. M. A.*, Jan. 28, 1911, Vol. LVI, No. 4, p. 259.

SECTION XI

SCHOOLS

It took a long time to realize that the whole child goes to school—his body, mind, and soul; that education of the mind alone is one-sided and may be hurtful; finally, that the hygiene of the child and his teacher, as well as the sanitation of school buildings and their equipment, is of fundamental importance. The combination of compulsory education and schools, having an unbalanced curriculum or impure water or vitiated air or improper sanitation, is nothing short of a crime by the state against the state. The child profits directly from attendance upon a school which has due regard for the child's physical well being and the development of his character; the state profits indirectly from the lessons in sanitation and hygiene which are carried into the child's home, and are applied as a matter of course in the home of the future citizen. Thus the principles of personal hygiene and sanitation become second nature, and in this way the conquest of the preventable diseases may be materially hastened. It is an economic waste to educate children and then permit them to die of some preventable infection before they have reached the period of maturity and productivity.

The school furnishes abundant material for the physiologist and the psychologist to study growth and development. The effect of the nature and order of the studies for each school year; the hours of work, rest, and play; the direction of physical exercise should all be regulated according to the average requirements and capacities of each school period, and should be based upon accurate observations extending over long periods of time. Both the immediate effects and the remote influences upon adult life should be taken into consideration. Youth is the time of unrest and activity, and it is part of the school work to direct these energies so as to obtain the best development; youth also requires generous nourishment and sufficient sleep. A child who comes to school tired and worn from disturbed slumber cannot profit in body or mind. The child who comes to school hungry or who does not have a judicious luncheon at the recess period is seriously handicapped physically and mentally. The quality of the food offered for sale at recess should be under close scrutiny. The hot lunches and nutritious food

furnished some of the school children in Boston and other cities at a reasonable price is a practical and wise movement.

One of the duties of the school is to teach and to require at all times cleanliness of person and clothing. The example of clean schoolrooms, corridors, lockers, toilets, basement, and grounds will, in time, influence the young citizen. Floors especially should be kept clean and the child be required to use the door mats before entering the building. Dust must be discouraged in all ways. In some schools in poor districts it is a good plan to have shower baths for those pupils who do not enjoy good bathing facilities at home. A toothbrush drill is the means of teaching many a child the first principles in dental prophylaxis. The teacher should be constantly on the lookout to impress upon the pupils the elementary facts in hygiene, such as turning aside the head and holding the handkerchief before the mouth and nose when coughing or sneezing. The teacher should discourage the habit children have of carrying their fingers to their mouths and noses. The anti-spitting rules should be reiterated and strictly enforced. The danger of mouthing toys and pencils and the habit generally of placing things in the mouth should be discouraged; "swapping" partly eaten articles of food should be prohibited, and the reasons explained. Cleanliness is not instinctive in children; it must be learned. The significance of modern biological cleanliness can come only through education and example. Progress in these matters cannot be made without an intelligent understanding on the part of the teacher. It is therefore important to teach the teacher.

Fatigue, prolonged and oft repeated, may injure the development and health of the child. Fatigue is favored by poor ventilation, compulsory sitting upon hard and ill fitting seats at improperly constructed desks, prolonged tension of a strict discipline, studies that are too intensive, and insufficient relaxation or inconsiderate treatment of the little ones. Discipline, obedience, and regard for the human rights of others are among the most important things learned at school. Many a child is unjustly disciplined and his little soul harassed through no fault of his own, but perhaps on account of defective eyesight or hearing, or other physical handicap, or some mental deficiency.

The question of home work should be carefully regulated in accordance with the capacity and age of the child. Children should not be kept busy at prescribed work most of the hours of the day. Some time should be left for quiet play and the encouragement of personal inclinations during which time the best development unconsciously occurs. Initiative, self-reliance, and self-help are submerged by lack of free time. The amount and nature of the work, both in and out of school, must be judiciously considered and should be based upon long years of careful study and observation. The immediate as well as

the remote effects should be taken into consideration. Many an ill-tempered child is simply overwrought and chronically tired out through excessive application of a conscientious and studious nature to tasks beyond the physiological capacity of his little brain and body.

The child should not be sent to school too young. Children must first learn to walk, run, talk, and coördinate muscles before they undertake reading, writing, and arithmetic. Pupils should not be graded according to their ages, but according to their capacity and physical development.

For the elementary schools one short morning session is enough. The general tendency is to reduce the hours of compulsory school attendance and increase the optional time through elective systems which encourage and foster native talents.

Primary pupils should not spend more than one-third of their school time in their seats. Exercises of various kinds that call into play muscular activity are most important at this age, not only for mental growth, but for physical growth, as well as for relief from the fatigue occasioned by sitting at desks.

The child on beginning school life enters an environment radically different from the free and active life which was his before school days began. The effect may be seen by the fact that children usually lose weight and the nervous system becomes affected during the first weeks of school.

Ungraded or special schools should be provided for backward and defective children and for those having favus, ring-worm, rachitis, or other conditions requiring either special pedagogical methods or particular medical treatment. Open-air, or fresh-air, schools for children who have or are threatened with tuberculosis serve a very useful purpose.

Finally, the whole school program should remember that the object is not to teach the child to be a child, but to direct his development so as to become a useful man or woman. The school system should therefore be carried out with due regard for future events and should be correlated with the adult life of the child.

School Building.—The school must be centrally located, so as to be convenient especially for the primary and grammar grades, and the school building should be modern, artistic, clean, and sanitary in all its appointments. Every school building should have playgrounds connected with it. Playgrounds should be level; about 30 square feet for each pupil is necessary to meet the demands of play. Thus 1,000 pupils require 300 x 100 square feet for playgrounds alone. In cities, roofs may be utilized for play. School-houses should be built in places that are quiet and free from nuisances, dangers of various kinds, and on ground that is either naturally dry or made so by subsoil drainage. The building should be solidly constructed and should stand apart, so that

sun and air may reach it from all sides. A substantial and artistic structure well placed has an important influence upon the young mind and character. Trees and judicious landscape gardening should provide shelter and shade and add to the attractiveness. The foliage, however, must not interfere with the light and ventilation of the school-rooms. If the building faces north, with corridors and stairs on this side, all the rooms will have sunlight at some time during the day. The best general arrangement of the plan of the building is that in which the school-rooms are all placed on one side of the building, with the corridors, halls, stairways, and wardrobes on the other. Built in the old way, with rooms around a central well, school-houses have dark central halls and staircases, and favorable lighting cannot be had in some of the school-rooms.

Buildings three or four stories high in schools which require the pupils to pass from the lower to the upper floors several times a day impose a stress in climbing so many flights of stairs that may be injurious to the pupils, especially to girls. Such buildings may be provided with elevators, or at least with inclines instead of stairs.

The basement should be under the whole building and carefully protected against dampness. Further, the basement should be well lighted and sunny.

School buildings should have at least two entrances, with doors opening outward; the halls and corridors should be generous and well lighted, and the stairs have easy risers and treads for children. The risers should be about 6 inches and the treads no greater than 12 inches.

The School-room.—The school-room is the unit in planning a school building; that is, the building should be a number of school-rooms properly disposed, and not a building cut into school-rooms whose size and arrangements are dependent upon the size and shape of the building.

Some of the important considerations in the school-room are the number of pupils to be accommodated, its size and shape, the amount and direction of the light, the ventilation and heating.

The minimum floor space for each pupil should be 15 square feet. If 18 square feet are allowed all exercises are made easier both for pupil and teacher. Two hundred cubic feet of air space is the minimum commonly allowed; therefore a standard school-room designed to accommodate 30 pupils should be 20 feet wide by 24 feet long, with a ceiling 13 feet high. The best shape for a school-room is that of an oblong, the width being to the length about as 3 to 4. No teacher should be required to have classes exceeding 30 pupils. The rooms, floor space, and air space should be at least as capacious for the primary as for the grammar grades.

The color of the walls should be such as to absorb the least light and prove least taxing to the eyes. A light green-gray is favored. The

surface should not be glossy and should either be coated with an oil paint, so that the walls may be washed, or, better, calcimined with a water paint that may be readily renewed. The ceiling should be white, so as to reflect the light.

The School Furniture.—The most important articles of school furniture, considered from the view of hygiene, are desks and desk chairs, for the reason that the pupil spends during school hours so much time at work at his desk. Unless, therefore, desks and chairs are constructed with full regard for certain well-known laws of hygiene they produce defects of eyesight, injurious effects as to posture, and wrong habits of carriage which are borne through life and, sadly enough, become more pronounced as the years increase.[1]

Professor Bowditch of Harvard University carefully measured and weighed 25,000 school boys and girls of Boston and found surprising variations. Taking ages on their last birthdays Professor Bowditch found the following variations in height:

	Boys	Girls
6 years of age	47.13[2] 40.66	47.36 40.57
Difference	6.47	6.79
11 years of age	57.50 49.47	57.96 49.33
Difference	8.03	8.63
15 years of age	67.90 56.55	65.00 57.39
Difference	11.35	7.61

[2] All figures are inches.

Besides the variations in height there is also variation in growth, and provision for this difference must therefore be made in the construction and adjustment of the desk and seat. The growth of girls is more rapid from 12 to 14 years of age, while boys grow most rapidly from 14 to 16 years of age. The annual growth during the maximum period is often an inch more than the annual growth at other periods. Further, there exist certain anatomical differences of proportion between boys and girls. The sitting height of girls is greater proportionately than their standing height in comparison with boys.

The Desk and Seat.—The desk and seat must therefore be adjusted so as to provide for differences of height and differences of growth. The desk must not be a prison stall, but should be comfortable and

[1] Shaw, Edward R.: "School Hygiene." The Macmillan Co., N. Y., 1902.

roomy. It must not favor the development of myopia and must not force a pupil into wrong postures. The matter is of greater importance than school men generally recognize.

The chair and seat should be of such a height that the thigh of the pupil when seated will be perfectly level, the lower leg being in an exactly vertical position, with the foot resting wholly upon the floor; that is, the thigh and the lower leg will, when the chair is of a proper height, form a right angle with each other. The seat must therefore be adjusted accordingly. The seat itself should not be flat, but somewhat

Fig. 139.—Faulty Posture. (Shaw's "School Hygiene," Macmillan Co.)

concave, the lowest part of the concavity being where the tuberosities of the ischium rest. The concavity has the additional advantage of counteracting the tendency to slide forward on the seat when the pupil leans back. The seat should have a back rest that will support the small of the back properly without leaning back excessively. Whether or not it supports the rest of the back is of small consequence. Support of the back carried to the level of the shoulder blades is likely to do more harm than good.

The distance between the seat and the desk should be such that the scholar may read at the desk and write on it without leaning forward more than a little and without entirely losing the support of the back

rest. The desk should not be so close as to press against the abdomen, nor near enough to interfere with easy rising from the seat. This means a distance of 10½ to 14½ inches from the edge of the desk to the seat back. It also means that the seat must not project under the desk more than an inch at most. The desk should be high enough for the arm to rest comfortably without much resting of the elbow; not, however, so low that the scholar must bend down to write on it.

FIG. 140.—THE HEUSINGER DESK. (Shaw's "School Hygiene," Macmillan Co.)

If the desk top is made to slide backward and forward it will give the pupil more freedom of movement while at the desk and will also permit him to sit down at the desk and rise from it with greater ease. One of the important considerations of a school desk is the proper slope of the top. It is well known that the line of sight which least taxes the eyes should fall upon the printed page perpendicularly to its

plane. To accomplish this some writers recommend a slope of 45° for the desk top; others 30°. These angles, however, are not practicable. The Vienna Expert School Desk Commission recommends an angle of 15° for the desk top, which is also approved by the experiments of Shaw. Such a slope permits a perfect posture in vertical writing.

A foot rest is sometimes attached to desks. The weight of opinion is now against foot rests, as they restrict the free movement of the pupil's feet while at the desk and interfere with opportunity to shift his feet and legs for relief from inactivity, and further interfere with the thorough cleansing of the floor under the desk. Shaw recommends the Heusinger desk, Fig. 140, and also the Ideal desk. The desk and seat shown in the accompanying photograph, Fig. 141, are known as the Boston school desk and chair. There are now many thousands in use in the Boston schools, and they are being adopted elsewhere.

Fig. 141.—Boston School Desk and Chair.

The seat and chair should be adjusted for each pupil when he enters school or is transferred to another room. Desks and seats should be adjusted at least twice a year: at the opening of school in September and again in February or March.

The Blackboard.—The blackboard should be placed upon the wall opposite the principal light. The board should not have a shiny, reflecting surface, and should never be placed between windows or near them.

The best blackboards are made of slate, as they can be washed, which lessens the dust nuisance. The best slate for this purpose is a greenish or strong black color, which is to be preferred to the grays and brownish-blacks. Colored crayons made with arsenic or sulphid of mercury carry danger and should be prohibited.

Posture.—Every condition must be eliminated and every care exercised to prevent the acquiring of physical defects in school, as well as to prevent the accentuation of those physical defects which the child may have possessed before entering school. Posture during sitting is of greater consequence than posture during standing, on account of the longer time the child sits and the muscular fatigue caused by the inactivity of a great number of muscles of the body for a long period.

Stooping over the desk leads to myopia; it also contracts the chest and interferes with free respiration, and puts additional labor on the heart; it results in round shoulders and curving of the spine backward and a carriage in which the head is pitched forward; it also tends to displacement of the internal organs, both of the abdomen and pelvis.

In order that the pupil may be in a proper physical condition to maintain an erect posture while in his seat, and thus form correct habits which he will carry through life, he must be given periods of relief from sitting at the desk and corrective exercises at different times during the day. In the first year the child should not be confined at his desk more than one-third of the time. In the succeeding years the total amount of time occupied at the desk may be gradually lengthened. In addition to the regular recesses there should be frequent short intervals of respite from sitting at the desk devoted mostly to some form of physical exercise.

A recess of not less than 20 minutes during the morning session and again during the afternoon session, when all pupils, if the weather and climate permit, go out of doors and engage in some form of active play, is of incalculable value in its results upon physical health and mental development. In addition there should be given to each grade every school day at least two short periods of systematic physical drills with the windows open.

Lighting.—The light must be of proper intensity, equally diffused, and come from the proper direction. So far as intensity is concerned the light must be neither too dim nor too strong, both extremes being harmful. The general rule is that the amount of transparent glass surface admitting light should be from one-sixth to one-fourth of the floor space. The correct amount of window space will depend on the location of the building, direction from which the light is admitted, size and shape of the room, and the proximity of other buildings or objects which might obstruct the light.

The amount of transparent glass surface required for proper illumination must be great enough to afford sufficient light on rainy, overcast, and cloudy days. Excessive window space is scarcely possible, for the excess illumination on bright days may be regulated and softened with shades and awnings.

The amount of illumination is measured by candle meters or candle feet; that is, the illumination afforded by a standard candle at a distance of one meter or one foot. Shaw[1] believes that the illumination should provide at least 50 candle meters in the most unfavorable part of the room.

Factory-ribbed glass or Luxfer prisms, which refract the light into the parts of the room where the light is needed, are a very decided ad-

[1] Shaw, Edward R.: "School Hygiene." The Macmillan Co., N. Y., 1902.

vantage, especially where schools have a small amount of free space in crowded city districts.

The principal light should come from the scholar's left, so as not to throw annoying shadows while writing. Windows may also be placed in the rear of the scholars. When practicable a skylight furnishes the best direction for illumination. Windows may also be placed at the right for ventilating purposes or for admitting direct sunlight while the scholars are not engaged in study. The window sash should be 3½ to 4 feet from the floor and should reach as near the ceiling as the construction of the building will permit, for the higher the windows reach the deeper the light penetrates into the room. Light should never enter from the front and shine in the pupils' eyes. Window curtains should be "opaque" and of a greenish cast. The upper fourth of the window furnishes one-third of the light, also the best light, hence it is obvious that curtains should not be hung from the top, but from the bottom, and should roll upward. Artists have long learned the lesson that light from above follows the direction of nature and is most agreeable and best.

Ventilation and Heating.—Ventilation of the school-room is of paramount importance. There is a great waste of time and energy of both the teacher and pupil working in a vitiated atmosphere, for pure air properly conditioned is necessary for mental work. Bad air means sluggishness, headache, listlessness, inattention, lack of energy, and a depression of mental vigor; further, bad air lowers resistance to certain diseases. In cold climates ventilation and heating go hand in hand.

In favorable climates and during mild weather the windows should be kept open. Even during cold weather the windows should be opened periodically and the room thoroughly flushed out with fresh air. The windows should always be thrown open at recess and also during calisthenic drills and physical exercises. The experience of the open-air and fresh-air schools teaches that cold is a fine tonic for mind and body.

Direct radiation from stoves or steam coils or hot-water pipes is inadvisable for school-rooms. The hot-air furnace may be used, provided the air is sufficiently moistened, but the direct-indirect system with steam or hot-water pipes is to be preferred. Two thousand cubic feet of air should be provided for each scholar hourly. The Massachusetts law requires 30 cubic feet of pure air every minute per pupil (1,800 cubic feet per hour). The fresh-air inlet should be capacious and separate outlets for the foul air should be provided. The cross-section of inlets and outlets should equal from 16 to 20 square inches for each scholar. Ordinarily it is preferable to place both inlets and outlets on the same side of the room, viz., upon the inner wall or warm

side. When so placed the warm air should be admitted about 7 feet above the floor and the foul air should pass out close to the floor.

Special attention should be given to the question of humidity, so that the warmed fresh air shall not be excessively dry.

The temperature commonly accepted as proper for a school-room is between 60° and 68° F. The children would probably work to better advantage if the temperature were kept a few degrees lower and the humidity kept so that the wet bulb never goes above 70° F. (see page 613. A thermometer should hang at about the breathing line in every school-room and the teacher should take hourly readings and keep a record. The temperature of school-rooms is usually too high, and those heated with the hot-air furnace are usually also too dry. Both extremes are prejudicial. If the air of the neighborhood is smoky and dusty it may readily be filtered before it is pumped into the school-room. The combination of the plenum and vacuum systems, the air being driven by rotary fans, is one of the best methods of artificially ventilating school-rooms. (See chapter on Ventilation.)

Water-closets and Urinals.—Separate accommodations must be provided for the sexes; privies in country districts should be in entirely separate buildings. The urinals should be constantly and automatically flushed and water-closets and urinals should be made to allow complete inspection and use of the scrubbing brush. Thorough ventilation of the toilet-rooms should be planned for and they should be kept clean and sweet at all times.

The water-closets may be in the basement if properly constructed and independently ventilated. The floors should be asphalted to facilitate cleaning and flushing, and should be scrubbed at least once a week. The toilet-room should be well lighted. Deodorizers should not be used, for if toilet-rooms are kept clean and water-closets well flushed they will not be necessary. Urinals should be made of slate or hard asphalt or other non-absorptive material, and one urinal should be provided for each fifteen boys. The outhouses in country schools should be properly constructed and under supervision. In fact, a matron should be in attendance to assist the little tots in the kindergarten and lower elementary grades, and a watchful eye on the part of the master of the school and those he delegates for this duty should be kept to prevent misbehavior in toilet-rooms.

Cloak-rooms.—There should be one cloak-room for each class-room, and it should connect both with the hall and the class-room. Cloak-rooms should be lighted from the outside, heated, and thoroughly ventilated to carry off odors and to dry the clothing. Hanging the clothing in the halls is undesirable, for obvious reasons. Each pupil should have a shelf on which to lay hats and small articles, hooks upon which to hang overcoats, and a space for rubber shoes and umbrella.

Teachers should see to it that the pupils do not sit in wet shoes and stockings or in wet clothes. Each school should have some provision for drying wearing apparel, such as a drying chamber which may be in charge of the janitor, to dry the wet clothing during school hours.

Dressing-rooms should also be provided for the teachers. All such rooms and lockers should be kept scrupulously clean.

Cleanliness.—Schools should be kept scrupulously clean and every precaution taken to prevent dust. Cleanliness of person and surroundings should be one of the important lessons which the pupil learns at school. Through example and discipline pupils should be taught to love order and neatness and to abhor untidiness and slovenliness. Cleanliness is the keynote of all sanitation.

Some of these requirements for schools are: clean drinking water; bubbling fountains and the abolition of the common drinking cup; discontinuance of the roller towel, cake of soap, brush, comb, or other toilet articles used in common; cleanliness of floors, desks, corridors, cloak-rooms, toilet-rooms, basement, and grounds; the prohibition of dry sweeping or dusting. Blackboards should be washed frequently to avoid the dust nuisance, and the floors may be treated with one of the dustless floor oils. The windows should be kept clean, and each child should have his individual books, pencils, and other accessories.

Medical Inspection of Schools.—The medical inspection of schools is no longer an experiment, but a pressing necessity. It is founded on a recognition of the close connection which exists between the physical and mental condition of children in the whole process of education. It seeks to secure ultimately for every child, normal or defective, conditions of life compatible with that full and effective development of its organic functions, its special senses, and its mental and spiritual powers which constitute a true education.

The object of a medical inspection of schools is not primarily the treatment of diseases, but rather their prevention. One of the principal objects is the early recognition of physical defects such as errors of refraction, imperfect hearing, malformations of the body from abnormal positions, adenoids, enlarged tonsils, and other obstructions of breathing, and sources of inflammation, etc. An important object of the medical inspection of school children is to determine their fitness to enter school and to recognize mental and nervous disorders; also the early recognition of the communicable diseases and measures to prevent their spread; the supervision of vaccination, and disinfection; the teaching of personal hygiene to pupils and teachers, and the sanitation and cleanliness of the school building and its surroundings; the adjustment of the seat and desk, and the medical supervision of the mental and physical work of the child.

Medical inspection of schools is making slow progress. A systematic

school inspection was started in Brussels in 1874 and in Paris in 1884, since which time the movement has become world-wide. In America the first systematic inspection of school children was begun in 1894, after four years' effort by Dr. Samuel H. Durgin, Commissioner of Health of Boston, who is regarded as the father of the system throughout America. The first scientific and extensive examination of school children was made by Dr. Henry P. Bowditch,[1] whose essay upon "The Growth of Children Studied by Galton's Method of Percentile Grades" has become a classic in the subject. In 1908 there were only seventy cities outside of Massachusetts having medical inspection of schools. Massachusetts has a compulsory medical inspection law; New Jersey has a permissive one; Vermont has a law requiring an annual testing of the vision and hearing of all school children, and Connecticut one providing for such tests triennially.

Physical defects are not equally significant either from the medical or from the pedagogical standpoint. Each kind of defect should be separately studied, and classifications should not include pediculosis with defective vision; club-foot with defective hearing; adenoids with ringworm.

The objects of the medical inspection of schools may be greatly assisted by teaching the teachers the elementary facts concerned.

Medical inspection of schools was organized in this country for the purpose of controlling the communicable diseases of childhood. It must at once be admitted that it has been a failure so far as this object is concerned, for it has had very slight influence upon the prevalence of measles, scarlet fever, diphtheria, whooping-cough, mumps, etc. Theoretically we would expect a good system of medical inspection of school children to check the prevalence of these diseases. Perhaps it does so to a limited extent. With improvements in the system and enthusiasm in the cause much may still be accomplished along these lines.

There has been much discussion concerning who shall conduct the medical inspection. It is plain that in any system the teacher must be the ultimate inspector, and teachers are quite competent to carry out simple tests for determining the acuteness of vision and hearing. In one sense the teacher is the foster mother of the child and frequently knows the child better than its own mother. The teacher should report to the medical inspector children who show any of the following symptoms: loss of weight, pallor, puffiness of the face, shortness of breath, swellings in the neck, general lassitude, growing pains, rheumatism, flushing of the face, eruptions of any sort, cold in the head, especially running eyes, irritating discharge from the nose, evidence of sore throat, cough, vomiting, or frequent requests to go to the toilet.

[1] Twenty-second Annual Report, State Board of Health of Mass., 1890, pp. 479-522.

The next most important link in the chain of a good system of medical inspection is the nurse. She is able to detect the beginning symptoms of disease and can be trusted to treat simple troubles. The chief value of the school nurse, perhaps, is in establishing communication with the home and securing friendly coöperation with the parents. Parental neglect is rarely due to the lack of parental affection, but to ignorance. The nurse is frequently able to gain the confidence of both child and parent when the medical inspector fails. The nurse, further, will assist the medical staff in carrying out treatment. One of the chief duties of the school nurse is a sort of social service.

It is the duty of the medical inspector to detect defects, not to treat them. Who shall treat the child is a matter for the parents or guardian to decide. It is not sufficient merely to notify parents that the child needs treatment, for frequently no attention is paid to the notices. The child may be referred to or taken by the school nurse to the hospital or outclinic. In some districts school clinics have been instituted with success.

Duties of the Medical Inspectors.—An ideal system of medical inspection of schools would consist of a corps of trained and competent physicians and sanitarians who would devote their entire time to this special work. The staff should have the assistance of experts in ventilation and heating, experts in sanitary architecture, experts in sanitary engineering, and experts in the various medical specialties.

Specialists should visit all school buildings no less than three times each year in order to investigate all matters of heating, lighting, and ventilation, cleanliness, gymnasiums, bath, and toilets, and the seating arrangements with reference to the size of the pupils; the purity of the drinking water, the quality of the food purchased by the children at the recess period, and the general conditions of the neighborhood that may affect the health of the pupils. Furthermore, coöperation between the medical and pedagogical departments should be helpful in solving the many difficult problems concerning the curriculum.

In addition to these general inspections all children entering school should be examined individually three times during the first year. The first examination is for the purpose of establishing whether the child is fit for school and can do the work without injury either to its mental or physical well-being. The second should be a physical examination, which may be made more thorough if the child is required to strip. This, however, should not be done unless the parents of the child are present or give their consent. The third examination consists of special tests of the eyes, ears, nose, throat, and teeth.

Aside from these regular examinations the school physician must respond to every call when a pupil comes to school having an eruption, fever, or other symptoms indicating a communicable disease. The med-

ical inspectors should also oversee disinfection, vaccination, and certify the return to school of any child who has been out of school by reason of a communicable disease.

The Communicable Diseases of Childhood.—Parents naturally come to regard the school as a veritable pesthouse for the spread of the communicable diseases of childhood—especially measles, whooping-cough, mumps, diphtheria, scarlet fever, chicken-pox, common colds, etc. Many of these diseases prevail in epidemic form during the summer time, when school is closed, and under other circumstances which show that epidemics may be independent of school attendance. It is difficult to determine just what part is played by the commingling of the pupils in school in the spread of such diseases and what part is due to other factors. Some diseases take a sudden jump in the autumn with the opening of school. Further, these diseases are not contracted by the school children alone, but are carried home to the other members of the household, and thereby create secondary foci. This problem of the communicable diseases and the schools is far from solution; the spread of these diseases has not been conquered by medical inspection, and their relation to school attendance is one that needs careful observation and study.

THE PERIOD OF EXCLUSION FROM SCHOOL FOR THE COMMON COMMUNICABLE DISEASES

Scarlet fever	6 weeks, or longer if redness of the throat, nasal discharge or other sequelæ persist
Measles	2 weeks from date of appearance of eruption
German measles	1 week from date of appearance of eruption
Chicken-pox	Until all scabs are gone
Diphtheria	1 week after second negative culture from nose and throat
Whooping-cough	8 weeks from appearance of characteristic cough
Mumps	3 weeks or longer if swelling persists
Pediculosis	Until all parasites and nits are gone
Ringworm Scabies Impetigo	Until examination reveals successful treatment

The question of closing the schools when some one of these diseases breaks out is often a difficult one to decide. If the children commingle out of school, upon the streets and playgrounds, no useful purpose is accomplished by closing the schools. At the beginning of an outbreak of measles or scarlet fever the schools may be closed for two weeks and then opened, but careful guard must be exercised to discover new cases

and a watch kept over the return of convalescents. Under these circumstances a daily inspection should be conducted before, and not after, the children enter school. If closing the schools for two weeks is not effective probably nothing will be gained by prolonging the period.

The diseases for which children should be excluded from school are: smallpox, scarlet fever, measles, German measles, chicken-pox, diphtheria, tonsillitis, whooping-cough, pediculosis, mumps, scabies, trachoma, ringworm, impetigo contagiosa, venereal disease, pulmonary tuberculosis, influenza.

The Eyes. —Errors of refraction are exceedingly common, and if not corrected are the cause of headache, nervousness, reflex pains, and a great variety of symptoms. They are also a great handicap to the mental and physical development of the child. The vision of all children should be examined annually, and at least once for color-blindness. It has been shown that the unnatural strain of accommodating the eyes to close work (for which they were not intended) leads to myopia in a large proportion of growing children. Thus the percentage of myopia increases markedly from the primary classes through the grammar grades, and is highest in the high-schools. The eyes should therefore be tested and errors of refraction corrected at least once a year. There are certain children who show normal vision by the ordinary tests (Snellen test type), yet whose eyes should be examined by an expert if they habitually hold the head too near the book (less than 12 to 14 inches); or if they frequently complain of headache, especially in the latter portion of school hours; or if one eye deviates even temporarily from the normal position. The following symptoms also indicate trouble with the eyes, viz., scowling and wrinkling of the forehead when reading or writing, twitching of the face, inattention, and slowness in book studies in a child otherwise bright.

The conditions which are especially hard upon the eyes are dim light, improper angle of vision, small print, and prolonged focusing at close range. Type for books should not be smaller than the following:

	Type	Width of Leading
First year	2.6 mm.	4.5 mm.
Second and third years	2.0 mm.	4.0 mm.
Fourth year	1.8 mm.	3.6 mm.
Above this grade	1.6 mm.	3.0 mm.

In addition to the size the characters should be simple, the ink black, and printed upon paper with a mat, unreflecting surface that is free from gloss. Paper of a gravish tone is to be avoided and the paper should be thick enough or of such quality that the print does not show

through from the back. Pupils should be taught that it is advisable while reading or during other close focusing of the eyes occasionally to look away and accommodate for distance to relieve the tension and counteract the tendency to myopia.

The Ears.—It has been found that approximately 20 per cent. of school children possess some defect of hearing either in one or both ears. Defective hearing is frequently mistaken for inattention upon the part of the pupil, for which he may be unjustly punished. Practical tests to determine the acuteness of hearing should be made separately with each ear by the use of a watch or by the whisper voice. Discharges from the ears, known as abscesses in the ears, or earache should at once be reported to the proper medical attendant.

The Teeth.—The proper use of the toothbrush and silk floss to keep the surfaces and spaces between the teeth clean should be impressed upon every pupil. For young children silk floss is not advisable if the space between the teeth is filled with soft tissue. The teeth should be examined by a competent dentist at least once, and preferably twice, a year. In the light of our present knowledge it is an outrage to allow caries of the teeth to develop into toothache before children are taken to a dentist. Irregularities of the teeth, especially those which make it impossible to close the mouth properly, lead to faulty digestion, to mouth breathing, and other defects. The first permanent molars are perhaps the most important teeth in the mouth, and are the most frequently neglected because they are so often mistaken for temporary teeth. It should be known that decay of the teeth is caused primarily by the fermentation of starchy foods and sugars, so that the greatest factor in preventing dental caries is the removal of food particles by frequent brushing and the use of the silk floss. Children should be discouraged from eating crackers and candy between meals and the teeth should be cleaned after each meal.

To provide expert dental attention for all carious teeth, including the temporary set, would overtax the facilities of any community. Dental clinics should be provided in which caries of the temporary teeth should have at least temporary treatment. It should be remembered that one infected tooth is like a rotten apple in a barrel in that it is apt to involve the others.

Nose and Throat.—The noses and throats of all pupils should be examined for any cause of obstruction to respiration, particularly adenoids, polypi, deviation of the septum, etc. Nosebleed should always be reported and inquiry should be made as to mouth-breathing during sleep. In all cases of acute illness the throat and mouth should be examined for indications of scarlet fever or measles and for the signs of tonsillitis or diphtheria, and a culture should be taken in any suspected case of diphtheria. The presence of a discharge from the nose

should be noted, and if it is thick and creamy a culture should always be taken. If the discharge from the nose is only from one nostril a foreign body or local cause should be looked for. Adenoids may be inferred from mouth-breathing, snoring, chronic post-nasal catarrh, or recurring ear trouble. Pupils with obviously large tonsils, recurring tonsillitis, and enlargement of the glands of the neck should be referred to a physician for treatment.

Diseases of the Skin.—Apart from the exanthemata the diseases of the skin which are of importance because communicable are: scabies, pediculosis, ringworm, and impetigo.

Scabies.—All children who are scratching or have an irritation on the skin should be examined for scabies (the itchmite). It is important that all infected members of the family be treated until cured, else the disease is passed back and forth from one to another. It is also important that all clothing, bedding, towels, etc., and similar things that come in contact with the body be boiled each time they are washed. All cases of scabies should be excluded from school until cured. Sulphur ointment is usually efficacious.

Pediculi Capitis.—Pediculi capitis (head lice) are extremely common among children, and are communicated directly and also by wearing each other's hats or hanging them on each other's pegs, or from combs and brushes. No person should be blamed for having lice, only for keeping them. The condition may be suspected by the teacher in children who show indications of irritation of the scalp, and the condition is easily detected by looking for the eggs (nits), which are small white objects adhering to the hair. Head lice are best treated by killing the living parasites with crude petroleum and then getting rid of the nits. With boys this is easy; a close haircut is all that is needed. With girls a fine-tooth comb wet in alcohol or vinegar, which dissolves the attachment of the eggs to the hair, may be used. All combs and brushes used should be carefully washed and disinfected. Children with pediculi should be excluded from school until their heads are clean.

Ringworm.—Ringworm of the skin yields readily to treatment, but upon the scalp is extremely chronic. When the disease attacks the scalp the hair falls off or breaks off near the scalp, leaving areas the size of a dime or dollar nearly bald. The scalp in these areas is usually dry and somewhat scaly, but may be swollen and crusted. The disease spreads at the circumference of the area and new areas arise from scratching, etc. The diagnosis is made by looking for the fungus.

Favus.—Favus is a disease somewhat allied to ringworm, more common in Europe than in America. In this disease quite abundant crusts of a yellowish color are present when the process is active. The roots of the hair are killed by the *Achorion schönleinii,* so that loss of hair

from this disease is permanent, a scar remaining when the condition is cured.

Children with ringworm or favus should not be allowed to attend school. Children should be taught to use their own brushes and combs and not to wear each other's hats, caps, etc. In some districts special schools are maintained for favus and for ringworm of the scalp, where the pupils receive treatment.

Impetigo.—Impetigo is a disease characterized by pustules which appear on the face, neck, and hands, less often upon the body and scalp. The size of the pustules varies very much and they often run together to form on the face large superficial sores covered with thick, dirty, yellowish, or brownish crusts. The disease is contagious and spreads by scratching as well as by using common towels and other things. Children having impetigo should not be allowed to attend school until all the sores are healed and the skin smooth.

Nervous Diseases and Mental Defects.—A sharp lookout for indications of diseases of the nerves and of mental defects should be kept and especial notice taken of suggestive symptoms in a child who did not formerly show them. The teacher should be taught to report instances of restlessness or inability to stand or sit quietly in a previously quiet child, especially if to this are added irritability of temper and loss of self-control, such as crying for trifles or inability to keep the attention fixed.

Chorea.—Twitching of the muscles, the result of disease, may cause the child to drop things, render his work awkward, or interfere with writing or drawing. Such children are too often scolded for being inattentive or careless. The indications of chorea (St. Vitus's dance) should not be confounded with habit-spasms such as blinking of the eyelids or the slower twitching movements of the face or shoulders or other parts of the body, which may be due to defects of vision, adenoid growths, or other reflex causes. Cases of chorea should be removed from school at once, both for the child's sake and to prevent an epidemic of imitative movements such as sometimes occurs. Children with habit-spasms need not be withdrawn from school work, although these conditions often require treatment.

Epilepsy.—Mild epileptic attacks (*petit mal*) are frequently overlooked or misunderstood by the teacher. They may be mistaken for fainting. Usually these attacks are only momentary, in which the child stares fixedly and does not reply to questions or in which he suddenly stops speaking or whatever he is doing and is unaware of what is going on about him. The lapse of consciousness is one of the characteristic features of epilepsy. The attack may be accompanied by rolling up of the eyes, drooling, or unusual movements of the lips; an epileptic fit often appears like a choking attack. Teachers very frequently mis-

understand epileptic attacks and cannot be expected to distinguish them from hysterical convulsions and other diseases. It does not necessarily follow that cases of epilepsy should be withdrawn from the school, but medical advice should always be had.

Neurasthenia.—Neurasthenia or nerve fatigue may be shown by irritability or sleeplessness and other indications threatening a nervous breakdown. This may be due to irregular habits, want of proper sleep, lack of suitable food, poor hygienic conditions, or simply from the child being pushed in school beyond his physical or mental capacity. Excessive fear or morbid ideas, bashfulness, undue sensitiveness, causeless fits of crying, morbid introspection, and self-consciousness may also be symptoms of a neurasthenic condition, and call for investigation and for the teacher's sympathy and winning of the child's confidence to prevent developments of a more serious nature.

The teacher should know that forgetfulness, loss of interest in work and play, desire for solitude, untidiness in dress or person, and like changes of character are sometimes incidental to the period of puberty.

Defectives.—Mentally defective children in the public schools exhibit certain common characteristics which soon become evident. The typical incorrigible child of the primary grades often is a mentally defective child of the excitable type. They are destructive, cruel to smaller children, and often precocious sexually. Certain cases show marked moral deficiency. Mentally defective children must be distinguished from those who are only temporarily backward as a result of some removable cause such as defective vision, impaired hearing, adenoid growths, or as the result of unhappy home conditions, irregular habits, want of proper sleep, lack of suitable food, bad hygienic conditions, etc. Teachers should refer to the medical inspectors for examination children who, without obvious cause, such as absence or ill health, show themselves unable to keep up in their school work, who are unable to fix their attention, or are incorrigible.

A careful lookout should be kept for children showing sexual perversion, for one sexual pervert may demoralize a whole school.

Vaccination.—Vaccination should be required of all children before they are permitted to attend school. The evidence of a successful vaccination usually accepted is a physician's certificate or a characteristic scar. For the indices of a successful take see page 11. School children should be vaccinated before entering school and again before entering high school.

REFERENCES

The following references have been used in the preparation of this chapter, especially the books of Shaw and Hogarth.

Shaw, Edward R.: "School Hygiene." The Macmillan Co., N. Y., 1902.

Hogarth, A. H.: "Medical Inspection of Schools." Oxford Medical Publications, London, 1909.

Gulick, L. H., and Ayres, L. P.: "Medical Inspection of Schools." New York Charities Publication Committee, MCMVIII.

Stevens, E. M.: "Medical Supervision in Schools." Baillière, Tindall, and Cox, London, 1910.

Baginsecky, Adolf: "Handbuch der Schulhygiene." Bd. I, 1898; Bd. II, 1900. Ferdinand Enke, Stuttgart.

Transactions of the International Congress on School Hygiene.

Zeitschrift f. Schulgesundheitspflege.

Bowditch, H. P.: "The Growth of Children Studied by Galton's Method of Percentile Grades." Twenty-second Annual Report of the State Board of Health of Mass., 1890, pp. 479-522.

Weyl, Theodor: "Handbuch der Hygiene." Vol. VII, Erste Abt, *Schulhygiene,* Jena, 1895.

SECTION XII

DISINFECTION

CHAPTER I

GENERAL CONSIDERATIONS

Definitions.—Disinfection means the destruction of the agents causing infection. An object is said to be infected when contaminated with pathogenic microorganisms. It is disinfected by destroying these organisms, whether they are in the substance or on the surface of that object. Disinfection, then, deals only with destroying the vitality of those minute forms of life which cause disease. It does not mean the destruction of all the lower forms of animal and vegetable life that may be in or upon an object—this is sterilization.

Sterilization.—All processes which sterilize necessarily disinfect, but all disinfecting processes by no means sterilize. The distinction between disinfection and sterilization arises principally from the fact that spores have a much greater resistance to all influences which destroy the vegetative cells. Fortunately, none of the pestilential diseases of man which occur in widespread epidemics, so far as known, are caused by microorganisms with resistant spores; therefore the usual processes of disinfection may be thoroughly efficient, yet leave many harmless and hardy forms of microscopic life alive. In other words, sterilization is rarely necessary in public health work, except in the case of anthrax, tetanus and other spore-bearing infections.

Antiseptics.—Antiseptic substances prevent decomposition and decay. Such substances retard or prevent the growth and activity of microorganisms, but do not necessarily destroy them; that is, antiseptics delay or prevent fermentation and putrefaction without destroying the microorganisms which cause these processes. There is a great difference between the antiseptic and the disinfecting power of most substances. For instance, a solution of formalin will restrain the development of most bacteria in the proportion of 1 to 50,000, but it requires a 3 to 5 per cent. solution of this liquid to kill the bacteria in a reasonably short time. As weak a solution of bichlorid of mercury as 1 to 300,000 will sometimes

prevent the germination of anthrax spores, whereas it requires a 1 to 1,000 solution to destroy them. Saturated solutions of salt or sugar will preserve meat, vegetables, and other organic substances; that is, they are antiseptic in their action but not germicidal, as they have small powers of destroying microorganisms.

Asepsis.—Asepsis means freedom from or absence of living microorganisms and is practically equivalent to sterilization.

Germicide.—A germicide is a substance or agent which destroys germs. Germicides and disinfectants are interchangeable terms, as both are used to indicate the destruction of microorganisms. Most germicides used in public health work are potent enough to sterilize objects with which they come in contact.

Deodorant.—A deodorant is a substance which has the power to destroy or to neutralize the unpleasant odors arising from organic matter undergoing fermentation or putrefaction. Such substances must be distinguished carefully from disinfectants. Deodorants destroy smells; disinfectants destroy germs. Many of the disinfecting agents are also deodorants, but all deodorizing substances are by no means disinfectants. For example, charcoal will absorb the malodorous gases arising from putrefying and fermenting materials, but it is inert so far as its power to destroy the cause of these processes is concerned. Formalin, on the other hand, is a true deodorant and disinfectant, as it combines with the organic matter to form new compounds which are both odorless and sterile. Bichlorid of mercury, while a very potent germicide, has practically no immediate effect upon odors. The volatile oils and other substances having a pungent odor are not deodorants; they simply cover up one smell with another.

Nature's Disinfecting Agencies.—In nature many forces are constantly at work to destroy infection and thereby limit the spread of the communicable diseases. It is the duty of the sanitarian to encourage the use of these natural disinfecting agencies; they are dilution, sunlight, dryness, and symbiosis. Sunlight is a great destroyer of germ life. Few microbes, especially the pathogenic ones, can live in the direct bright sunlight many hours. Dryness is another natural condition that is destructive to many of the minute forms of life with which we have to contend. The combination of dryness and sunlight is quite as good, if not better, than the ordinary fumigating processes which are commonly used in practical disinfection against surface contamination. Dryness, sunlight, and cleanliness are the keynotes of sanitation in the modern acceptation of the term.

We now know that most of the pathogenic microorganisms do not grow and multiply in our environment. For the most part they die when wafted into the air or carried into water or deposited in the soil. It is only occasionally that they find conditions favorable for

development in foods such as milk and meat, and exceptionally in water. Further, it is to be noted that ordinarily it requires a certain number of microorganisms to produce infection. It is quite conceivable that a single typhoid bacillus or a single tetanus spore may "kindle a conflagration." Experimental evidence with the infections upon laboratory animals teaches the lesson that ordinarily an animal is capable of taking care of minute and dilute amounts of infection. Dilution, attenuation, and the conditions of our environment, unfavorable to most germs harmful to man, therefore protect us in no small measure against the communicable diseases.

Cleanliness.—Cleanliness is a very important adjunct to the work of disinfection. In fact, cleanliness lies at the base of all our sanitary measures. The mere act of cleaning removes some of the adherent microbes from the surface and the ordinary scrubbing and washing result in the final destruction of many more. Dry dusting and sweeping serve only to stir up dust and infection, which settle down again upon the same or other surfaces. Cleanliness serves another important purpose, so far as infection is concerned; it removes the organic matter on which and in which bacteria may find favorable conditions for prolonging life and virulence. The modern conception of cleanliness has expanded with the growth of the sanitary sciences. We now aim at biological cleanliness as well as esthetic cleanliness. This includes not only the removal of organic matter, but the destruction of insects and vermin, and their feeding and breeding places. So far as personal cleanliness is concerned, the two important acts to prevent infection are: (1) Washing the hands before eating and (2) keeping the fingers away from the mouth and nose.

In the wholesale disinfection which must be practiced to check widespread epidemic diseases due to bacterial infection we are largely limited to the use of the agents which nature has constantly at work to destroy such infection. Against a single case of communicable disease or against a limited infected area we may employ aggressive measures such as steam and strong chemicals; but when a disease, due to bacterial infection, has spread over an extensive district these methods must be supplemented by all the resources of nature. The people must be educated so as individually to employ intelligent measures to avoid the infection. Cleanliness must be more scrupulously practiced than ever, sunlight and dryness must be given their fullest opportunity to operate even at the expense of a few faded carpets or colors.

Symbiosis.—Many pathogenic microorganisms are destroyed in the process of putrefaction and fermentation. They die in the fierce struggle for existence going on in these processes of decomposition. For the most part the hardier saprophytic forms of life overpower and kill the disease-producing microorganisms having comparatively feeble powers

of resistance. The fact that infected carcasses, sewage, and putrid organic matter generally purify themselves by the very processes that destroy them is a fortunate provision of nature.

When and Where to Disinfect.—It naturally suggests itself that it is much better to prevent infection than to be compelled to destroy it after it has become disseminated through ignorance, carelessness, or negligence. It is the duty of the disinfector to destroy infection whenever it is found; it is the ideal of the sanitarian to prevent the spread of infection so as to render broadcast disinfection unnecessary.

The best place to apply disinfection is at the seat of origin of the infection. Man is the fountain-head of most of the infections to which he is heir; hence the most effective place to apply disinfectants is at the bedside, and to the excretions, especially those from the mouth, nose, and bowels. When proper precautionary measures have been taken at the bedside with a case of cholera, typhoid fever, or plague there is little need of subsequently disinfecting the sickroom, but when a diffusion of the infection results then a general disinfection becomes necessary.

Qualifications of the Disinfector.—The disinfection of any given place is a complex operation, and should not be attempted by anyone not familiar with the peculiarities of the particular infection with which he has to deal and a thorough knowledge of the disinfecting agents employed. In other words, it is quite as important to know *what* to disinfect as *how* to disinfect and *when* to disinfect. A thorough understanding of the causes and modes of transmission of the communicable diseases is the most useful weapon the disinfector has in his fight against the spread of infection.

The success of the disinfector lies in personal attention to minute details. Germs are little things, and it is little things that count in this kind of work. The disinfector who is satisfied to leave the process in the hands of an inexperienced person with a few words of instruction cannot expect to obtain trustworthy results. The disinfector must give personal surveillance to the whole process—the materials, the strength of solutions, modes of application—and must be present to guide and direct every step of the operation with the same conscientiousness and thoroughness with which the surgeon assures himself of every detail of asepsis in his operating-room.

Controls.—Every disinfecting process should be controlled by exposing cultures upon preparation slips or threads as a guide and check to the thoroughness of the process. This may perhaps best be done by saturating threads with an active culture of *B. prodigiosus*. These threads are attached to little slips of paper which are then exposed in various portions of the room to be disinfected. After the completion of the operation the threads are inoculated into Dunham's peptone

medium. If the *B. prodigiosus* has survived the characteristic red color appears in the culture medium.

Disinfection Must Be in Excess of Requirements.—The disinfection of rooms, bedding, ships, and objects that have been exposed to infection must of necessity be greatly in excess of the actual requirements. This is one of the difficulties met with in attacking an invisible foe. A sickroom might readily be disinfected and rendered safe by applying a few gills of one of the germicidal solutions to a small spot or a limited area. But, as we cannot see the germs, it is necessary to apply our disinfecting agents to every inch of surface of the room and all its contents in order not to miss that particular spot. At first disinfection was directed by a shotgun process in a general sort of blunderbuss way against everything, but now that we know more about the habits and habitat of each one of the particular microorganisms we can concentrate our efforts with some exactness upon the particular object liable to transmit infection, and with greater assurance of eradicating danger.

The Ideal Disinfectant.—The ideal disinfectant must first and foremost possess a high germicidal power. It must not be handicapped by the presence of organic matter; it must be reasonably stable, so as not to deteriorate under ordinary conditions; it must be soluble or readily miscible in water; if it forms an emulsion the emulsion should be permanent; it should be harmless to man and the higher animals; it should have the power of penetration; it should not corrode metals, bleach pigments, or rot fabrics, and, finally, it should be reasonable in price.

The stress of modern activities demands disinfecting processes that are instantaneous in their action, all-pervading in their effects, cheap, harmless, and free from unpleasant odors that might be offensive to the fastidious. Such perfect disinfectors are not known. It requires money and the expenditure of well-directed and intelligent energy to accomplish satisfactory disinfection. No one substance is applicable to all diseases or to all substances, or even to the same disease or the same substance under different conditions.

Terminal Disinfection.—Terminal disinfection during recent years has been disparaged as a public health measure because it has little effect upon the control of the communicable diseases and the cost of such disinfection appears to be disproportionately large to the benefits. The evident limitations of terminal disinfection have cast doubt in the minds of some health officers upon the value of disinfection in general. This is an unfortunate attitude. No one can question the great value of disinfection properly applied. It is, of course, much more important to destroy the infection in the discharges throughout the course of a case of typhoid fever than to trust to one final disinfection of the sickroom and its contents. The same holds with about equal force for most of the communicable diseases. We now know that fomites play a com-

paratively minor rôle in the transmission of disease. The disinfection of rooms and objects does not now, therefore, hold the importance in the minds of sanitarians that it once did. However, if terminal disinfection prevents the occurrence of only a small number of cases it would still seem to be worth while. Moreover, what health officer would willingly allow his child to occupy the bed or handle the objects in a room soon after a case of typhoid, scarlet fever, tuberculosis, or diphtheria without first applying some effective method of purification? The greater the care and cleanliness exercised during the progress of the disease the less the need of terminal disinfection. So long as we possess such a reasonably efficient and satisfactory substance as formaldehyd, terminal disinfection should be practiced after all diseases in which the environment may become infected, even though the danger be slight.

The Standardization of Disinfectants.—There is no accurate standard by which the power of disinfecting agents may be measured. There are conditions influencing the life of the bacterial cell which we are unable to control. It is for this reason that the strengths of solutions necessary to disinfect are variously stated by different authorities, and the time of exposure is for the same reason not always definitely decided. The difficulty in this connection is to determine the minimum conditions which will furnish trustworthy results and still provide a coefficient of safety necessary for general practice.

While the results of scientific work in the laboratory must be our guide as to the value and efficiency of any disinfecting process we cannot ignore the results of experience gained in actual practice in combating the communicable diseases. This is especially true of disinfectants used against a disease the cause of which is only surmised or the mode of transmission not definitely known. We have had a lesson on this point in the case of sulphur. This substance had long been used as a disinfectant for yellow fever, and practical experience had justified the confidence placed in sulphur fumigation to check the spread of this disease, but when the scientific tests made in the laboratory showed that sulphur dioxid is a very poor germicide discredit was thrown upon it; now that we know that sulphur dioxid is one of the best insecticides confidence has been restored both as to the scientific and practical value of this substance.

On the other hand, laboratory experiments have established with great accuracy the value and reliability of certain disinfectants which otherwise would have gone begging. Some substances, such as zinc chlorid and sulphate of iron, have been robbed of the high value in which they were formerly held and placed near the bottom of the list of disinfectants. Even carbolic acid has been shown to have less germicidal power than was supposed.

Methods.—No satisfactory method of estimating the comparative

germicidal value of disinfectants under the varying conditions met with in actual practice has yet been devised. Some of the principal factors to be reckoned with are the character of the microorganisms to be destroyed, the nature of the medium in which they exist, the temperature at which the disinfecting process is carried on, the time during which the disinfectant is allowed to act, its chemical nature, power of penetration, etc., etc.

Koch[1] in 1881 used cultures of *B. prodigiosus, B. pyocyaneus,* and *B. anthracis,* both with and without spores. He soaked threads in a culture of the test organism and afterward dried them for various periods and then exposed these infected threads to the action of the disinfectant to be tested. The threads were then washed and laid on the surface of a solid nutrient medium and incubated for growth. This method, although characterized by greater scientific accuracy than the methods previously used, lacked perhaps those broader features of the older, rougher experiments; that is, the method did not approximate the conditions met with in practical disinfection closely enough.

G. Simms Woodhead[2] in 1887 used silk threads which, after being thoroughly dried, were soaked in a culture or emulsion of the microorganism. These threads were placed in the disinfectant to be tested, then thoroughly washed in distilled water, and transferred to fluid nutrient medium. Similar methods were used by Fraenkel,[3] Behring,[4] and many others.

Stenberg[5] in 1888 described a method which he used as early as 1880. He mixed 5 c. c. of a young culture with equal quantities of the solution of the germicidal agent. Thus 5 c. c. of a 1 to 200 solution of carbolic acid would be added to 5 c. c. of a recent culture of typhoid, and after stated intervals 1 or 2 loopfuls would be transferred to a nutrient medium. This was evidently the predecessor of the drop method.

The next modification was to add a small quantity of the bacterial emulsion to a large quantity of the disinfectant, thus reducing to a minimum the amount of foreign matter in the mixture.

Krönig[6] and Paul in 1897 adopted an entirely original plan. They coated small garnets of uniform size with an emulsion containing sporulating anthrax bacilli. These were dried and then dropped into the

[1] *Mitteilungen aus dem kaiserlichen Gesundheitsamte*, I, 1881, abstracted by Whitelegge, in "Recent Essays," New Sydenham Society, London, 1886, Vol. CXV, p. 493.

[2] *Proceed. of the Roy. Physical Soc.*, Edinburgh, 1887, Vol. IX, p. 386.

[3] "Die Desinficerenden Eigenschaften der Kresole," *Zeit. f. Hyg.*, Leipzic, 1890, Vol. VI, S. 521.

[4] "Ueber Desinfektion, mittel und-methoden," *Zeit. f. Hyg.*, Leipzic, 1890, Vol. IX, S. 395.

[5] "A Manual of Bacteriology," New York, 1893, p. 186.

[6] "Die chemischen Grundlagen der Lehre von der Giftwirkung," *Zeit. f. Hyg.*, Leipzic, 1807, Vol. XXV, S. 1.

disinfecting solution. After exposure for stated intervals the garnets were removed, rinsed, and the organisms washed off in sterile water, plated, and counted.

Rideal and Walker in 1903 introduced a method by which they

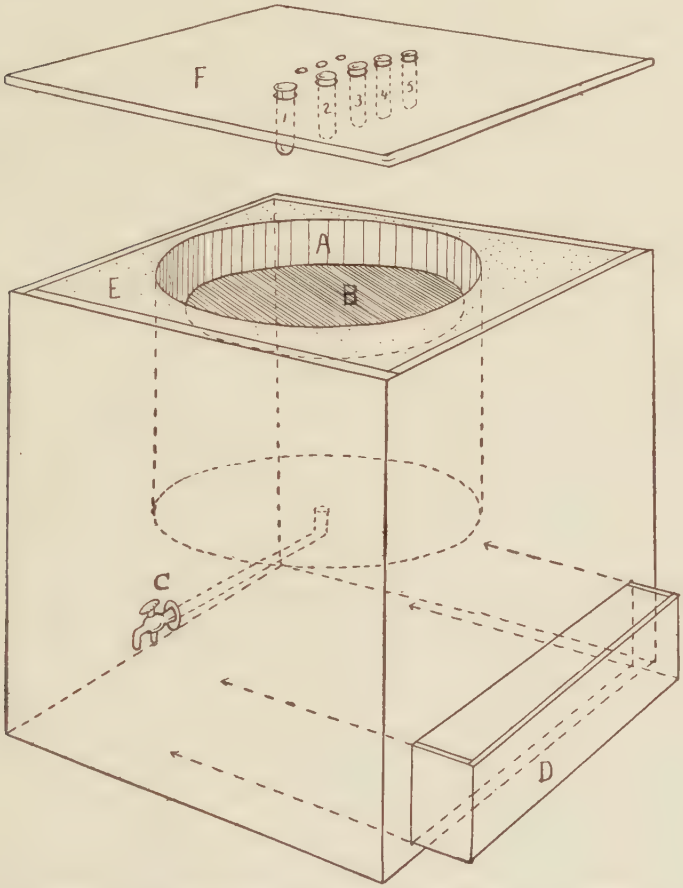


FIG. 142.—DEVICE FOR DETERMINING CARBOLIC COEFFICIENTS. Consists of a wooden box 14" long by 14" wide by 15" high, containing a metal pail (A) 10" in diameter, and 8¼" deep. A shelf made of wire mesh (B) is inserted 2" from the top of the pail, which is filled with water. A pipe with a faucet (C) from the bottom of the pail will be found very convenient to draw off the water and regulate its temperature. Asbestos packing (E) completely surrounds the pail in order to insulate it. The lid of the box (F), which is raised in the drawing, contains openings for the five test-tubes, and three other openings for cultures and thermometer. When the lid is in place the test-tubes rest upon the shelf (B). A drawer (D) in the bottom of the box is convenient to keep test-tubes, inoculating needles, thermometer, and other parts of the apparatus.

proposed to determine and state in definite numerical terms the value of any disinfectant. This they called the "carbolic-coefficient," for the reason that carbolic acid is taken as the unit or measurement against which the germicidal power of all other substances is compared.

The Carbolic Coefficient.—This test, sometimes known as the Rideal-Walker method of standardizing disinfectants, has been variously modified and improved.[1] It is at present the best method we have for comparing the strengths of germicidal substances in solution. The method, however, has distinct limitations, as it only gives information concerning the relative value of germicides upon the naked germ cells under favorable conditions of action.

In order to obtain results that may have comparative value and to avoid discrepancies it is of the greatest importance to keep all the factors of the test uniform and to give attention to every detail. The following are the more important factors and principles upon which this test is based:

Time.—The time is taken as the constant and the strength of the disinfectant as the variant. It is easy to demonstrate that, if reversed, totally erroneous results will be obtained.

Test Organism.—The culture recommended is a 24-hour-old *B. typhosus* grown in bouillon. It is important always to use the same strain of typhoid, as different races vary in resistance. Further, the culture should be carried over every twenty-four hours on at least three successive days before using it in a test. It is sometimes advisable to filter the culture through filter-paper in order to remove clumps just before beginning a test. The culture should always be grown under the same conditions, upon the same medium, so as to insure uniformity.

Medium.—The standard beef-extract broth (reaction + 1.5) recommended by the Committee on Standards of the American Public Health Association for Water Analysis, is used both to grow the test typhoid organism and also for the sub-cultures made after exposure to the disinfectant. Ten c. c. of this broth are placed in each test-tube for the sub-cultures, as this amount is sufficient to avoid any antiseptic activity of the disinfectant carried over.

Temperature of Exposure.—This is one of the most important factors. The germicidal activity of substances increases with the temperature. In this respect germicidal reactions resemble chemical reactions. It is therefore of the utmost importance that the solutions tested should be always at the same temperature, and for this purpose 20° C. has been selected. The solutions to be tested and the typhoid culture itself must be brought to this temperature before they are mixed, and then maintained at this temperature in a water-bath.

Proportion of Culture to Disinfectant.—Rideal and Walker first proposed to use one drop of the typhoid culture to each cubic centimeter of

[1] Rideal, S., and Walker, J. S. A.: *Jour. Roy. San. Inst.*, London, 1903, Vol. XXIV, p. 424. "The Standardization of Disinfectants," The Lancet Commission, Vol. CLXXVII, Nos. 4498, 4499, and 4500. Anderson and MacClintic: *Jour. Infect. Dis.*, Vol. VIII, No. 1, Jan., 1911, pp. 1-26.

germicidal solution. It is more accurate to use a measured amount, say 0.1 c. c. of the 24-hour-old bouillon culture of typhoid to 5 c. c. of solution. These are convenient amounts easily and accurately measured with standardized delivery pipettes. It should be kept in mind that the addition of the bouillon culture dilutes the germicidal solutions, but as this is a constant factor it does not affect the comparative values as expressed by the carbolic coefficient, but may be taken into consideration in judging the germicidal values for practical work.

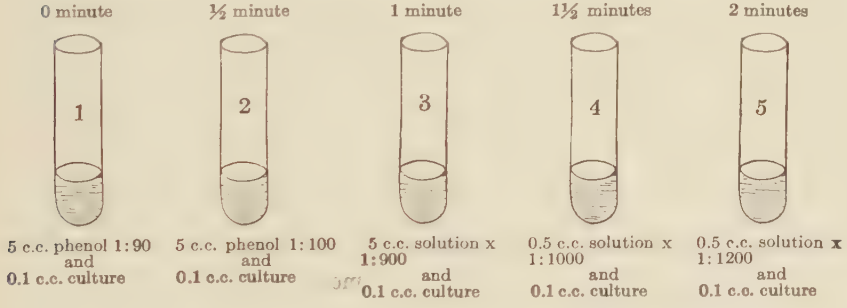
Inoculation Loops.—Precisely the same quantity of fluid from the mixture should be removed each time for the transplants. This is done most readily with platinum loops made of 23 U. S. standard gage wire and a loop 4 millimeters in diameter. Several of these loops should be on hand. They are sterilized and placed upon a rack. As one is used it is flamed and returned to the rack, so that it will be cool when taken in its turn.

The following method is the one used in my laboratory for carrying out the carbolic coefficient:

A solution of 5 per cent. phenol c. p. is made and standardized chemically.[1] The usual dilutions of 1 to 90, 1 to 100, and 1 to 110, etc., are made from this stock solution as desired.

The solutions of the germicidal substances to be tested must be made accurately, according to volumetric or gravimetric methods.

ARRANGEMENT OF THE TUBES IN THE WATER-BATH AND THEIR CONTENTS



The tests are carried out in test-tubes one inch in diameter and three inches long. These are placed in a row in a water-bath. The test-tubes rest upon a bed of sand and are held in place by a wire rack or simply by a board perforated with holes of suitable size. If the water-bath is sufficiently large and the water brought to just 20° C. it may be maintained at this temperature with but slight attention.

Each test-tube receives 5 c. c. of the solution to be tested. Time is

[1] By bromin titration. (See description in Sutton's "Volumetric Analysis.")

allowed for the solutions to reach the temperature of 20° C., then the culture which has previously been brought to 20° C. is added and mixed with the solution in each test-tube in turn. The culture is added to each tube at intervals of just 30 seconds. With a row of five tubes this will make a 2½-minute interval for each particular test-tube.

Two and one-half minutes after the phenol and the culture have been mixed together in tube No. 1 a loopful of the mixture is removed and planted in broth; 30 seconds later a loopful of the mixture is taken from tube No. 2, and so on throughout the series at intervals of 30 seconds. The entire procedure of removing the loopful of mixture and planting it into one of the test-tubes containing the beef-extract broth requires only about 15 seconds, allowing plenty of time to flame the loop, replace it in the rack, and pick up another loop which had previously been flamed and has cooled sufficiently for the next operation. The test-tubes holding the mixture of germicidal solution and culture need not be removed from the water-bath, and it is not necessary to keep them plugged with cotton. The loop should always be plunged to the bottom and care taken not to touch the sides of the test-tube and always to carry away a loopful of the fluid to be transplanted. The test-tubes holding the beef-extract media for the transplants are conveniently placed in wooden racks and are incubated at 37° C. for forty-eight hours, when the readings as to growth (+) or no growth (—) are tabulated.

An example of a carbolic coefficient test follows:

	2½ Minutes	5 Minutes	7½ Minutes	10 Minutes	12½ Minutes
Phenol 1 : 90	+	—	—	—	—
Phenol 1 : 100	+	+	—	—	—
Solution X 1 : 900	+	—	—	—	—
Solution X 1 : 1,000	+	+	—	—	—
Solution X 1 : 1,200	+	+	+	+	+

The carbolic coefficient of solution X is therefore $\frac{1,000}{100}$ =10.

Anderson and McClintic[1] have modified the procedure employed by the Lancet Commission to determine the coefficient to be the mean between the strength and the time coefficient; that is, the figure representing the degree of dilution of the weakest strength of the disinfectant that kills within 2½ minutes is divided by the figure representing the degree of dilution of the weakest strength of the phenol control that kills within the same time. The same calculation is done for the weakest strength that kills in 15 minutes. The mean of the two is the coefficient.

Kendall and Edwards[2] have devised an ingenious method to deter-

[1] *Hygienic Laboratory Bulletin*, U. S. P. H. and M. H. S., No. 82, 1912.
[2] *Jour. Infect. Dis.*, Vol. VIII, No. 2, March, 1911, pp. 250-257.

mine the penetrating power of germicides in the presence and absence of organic matter. The method consists essentially of cylindrical moulds of agar impregnated with the test organism. Sections of these cylindrical moulds or artificial feces are exposed to the germicide solutions and plants made after proper intervals of time from a core taken from the center of the cylinder.

Interpretation of Results.—A low carbolic coefficient means a useless disinfectant; on the other hand it should be remembered that because a germicide has a high carbolic coefficient is no true indication that it is a favorable agent in practical work. There are many factors still to be considered. Thus a useful disinfectant should not be very poisonous to higher animals; should not corrode metals or rot fabrics; should not stain or bleach; should not have an unpleasant smell; should be reasonably cheap; should be readily miscible with water and not deposit from solution or suspension; should be reasonably stable; should act both in alkaline and acid media; should not be greatly influenced by the presence of organic matter, and should possess a fair power of penetration. It must at once be evident that no one test can determine all of these factors, so that a thorough and comprehensive study of the substance to be used should be made upon many different parasites under many different conditions before we can have a satisfactory knowledge of its power and limitations. This is one of the reasons that makes us conservative about taking up new germicidal substances until thoroughly tested under different conditions, and inclines us to adhere to well-known chemicals such as bichlorid of mercury, carbolic acid, the coal-tar creosotes, lime, the hypochlorites, and formalin, the advantages and limitations of which have been thoroughly established.

THE PHENOL COEFFICIENT OF SOME COMMERCIAL GERMICIDES
Determined by Thomas B. McClintic[1]

	Without Organic Matter	With Organic Matter
Bacterol	1.58	1.34
Benetol	1.23	0.92
Cabot's Sulpho-Napthol	3.87	2.33
Carbolene	1.36	0.65
Carbolozone	1.48	0.48
Car-Sul	2.00	1.75
Chloro-Naptholeum	6.06	3.21
Cremoline	1.26	0.69
Creo-Carboline	4.03	2.26
Creolin-Pearson	3.25	2.52
Cresoleum	2.90	1.75
Crude Carbolic Acid	2.75	2.63
Dusenberry's Liquid Creoleum	1.00	0.40
Germol	2.12	1.79
Hycol	12.30	9.37
Hygeno A	3.56	1.81
Kreosota	1.26	0.65
Kreotas	1.10	0.30
Kreso	3.92	2.32
Kresolig	2.18	1.48
Lincoln Disinfectant	1.48	1.10
Liquor cresolis compositus (U. S. P.)	3.00	1.87
Lysol	2.12	1.57
Napthalene	2.50	1.36
Phenoco	15.00	9.86
Phenol liquid (U. S. P., 1890)	1.77	1.76
Phenosote	3.43	2.31
Phinotas	1.37	0.53
R. R. Rogers Disinfectant	3.03	2.05
Rudisch's Creolol	1.24	0.75
Saponified Cresol	1.03	0.57
Tarola	3.12	1.93
Trikresol	2.62	2.50
Zenoleum	2.25	1.64
Zodone	1.62	0.51
Zonol	2.37	1.57
Antozone[2]	nil	.....
Creola Disinfectant	0.52	.....
Dioxygen	weak	.....
Electrozone	0.90	.....
Formacone Liquid	weak	.....
Killitol	weak	.....
Kretol	0.92	.....
Listerine	weak	.....
Phenol Disinfectant and Cleansing Liquid	0.61	.....
Phenol Sodique	weak	.....
Pino-lyptol	0.27	.....
Platt's Chlorides	weak	.....
Public Health Liquid Disinfectant	0.48	.....
Sanitas	0.30	.....
The Twentieth Century Disinfectant	0.13	.....
Veroform Germicide	0.43	.....
Worrell's Insect Exterminator and Disinfectant	weak	.....
Zodane No. 3	weak	.....

[1] *Hyg. Lab. Bull. No. 82*, U. S. P. H. & M. H. S.

[2] The following disinfectants have a coefficient of less than 1. Most of them are so weak that it was impracticable to determine the coefficient.

CHAPTER II

PHYSICAL AGENTS OF DISINFECTION

Sunlight.—Sunlight is an active germicide. It destroys spores as well as bacteria. Unfortunately, the sunlight is so uncertain and the force of the sun's rays so variable and their disinfecting powers so superficial that it cannot be depended upon as an aggressive measure in attacking infection. In rooms, ships, and confined spaces sunshine comes more under the purview of the sanitarian than under that of the disinfector, but the latter can always use it to advantage in supplementing his other methods. Room and objects may always be sunned and aired with advantage after disinfection.

The different rays of light have very different effects upon germ life. The blue-violet and ultra-violets, that is, the more refragible chemical rays of short wave length, are the only ones possessing germicidal power. The red and yellow rays are practically inert in this regard. The source of light seems to have little influence upon the result; it is more a question of intensity and nature of the rays. Even diffused light retards growth and development of microorganisms, and if strong enough will finally kill them. Electric light containing the proper rays is efficient. The Röntgen rays have no bactericidal properties.

The time required for light to destroy bacteria varies with its brightness and with conditions such as moisture, temperature, transparency, and composition of the media, which aid or hinder the effect of the rays. The time also varies with the different microorganisms; plague bacilli and cholera vibrio usually die more quickly than tubercle bacilli. Spores are much more resistant to the influence of the chemical rays than the bacterial cells themselves. Thus it usually requires about 30 hours' sunning to kill an anthrax spore, while the anthrax bacillus is killed in one or two hours under the same conditions.

Ultra-violet Rays.—Ultra-violet rays obtained from the Cooper Hewitt mercury vapor lamp and other similar devices have an exceedingly powerful germicidal action, killing spores as well as bacterial cells. Glass is opaque to these rays of short wave lengths and it is therefore necessary to use quartz globes. This method has recently

come into use for the sterilization of water and other substances. (See page 801.

Electricity.—It appears that electric currents have little germicidal action in themselves and that the apparent effects noted by some investigators are due either to the heat generated by the current or to electrolytic action. Electricity has very little use in practice as a disinfectant. Hermite used the products of electrolysis for the sterilization of sewage. He added sea-water to the sewage and the electrolytic action caused the formation of hypochlorite, which has well-known germicidal action. The effect of electrical currents upon bacteria seems to be a purely chemical one in the case of antiseptic substances, being formed by electrolytic decomposition; or a thermal one in the case of the production of heat, which so frequently attends the discharge of electric currents.

Burning.—Fire is the great purifier. Burning has, however, a very limited range of usefulness in practical disinfection. The disinfector is seldom justified in burning an article against the wish of its owner, for we now possess methods by which any object may be rendered safe so far as its power of conveying disease is concerned. In actual practice, however, the disinfector often comes across a great amount of rubbish and articles of little value that he will find safer and cheaper to burn than to disinfect. The burning of garbage and refuse is the safest means of disposing of such organic substances from a sanitary standpoint, especially in districts where pestilential disease prevails. From the same standpoint the cremation of all bodies dead of a communicable disease is the safest method of preventing possible spread of infection from this source. Burning is the more satisfactory method of disinfecting and disposing of small amounts of sputum and other infected discharges. Burning of the surface of the ground by means of gasoline torches and petroleum is sometimes used to destroy animal parasites and other infections which find lodgment on the soil. The gasoline torch is also used to fight insect pests of trees and plants.

Dry Heat.—A temperature of 150° C. continued for one hour will destroy all forms of life, even the most resistant spores. It is easy to maintain this temperature in an apparatus of special construction known as a hot-air or dry-wall sterilizer. Glassware and many objects that will stand this degree of heat are sterilized in an oven of this kind in bacteriological laboratories and in surgical clinics. Dry heat is not as satisfactory a disinfectant as moist heat, as it lacks the power of penetration and is injurious to fabrics. Most materials will bear a temperature of 110° C. without much injury, but when this temperature is exceeded signs of damage soon begin to show. Scorching occurs sooner with woolen materials, such as flannels and blankets, than with cotton and linen. Over-drying renders most fabrics very brittle, but

this injury may be lessened by allowing the materials which have been subject to dry heat to remain in the air long enough to regain their natural degree of moisture and pliability before manipulating them.

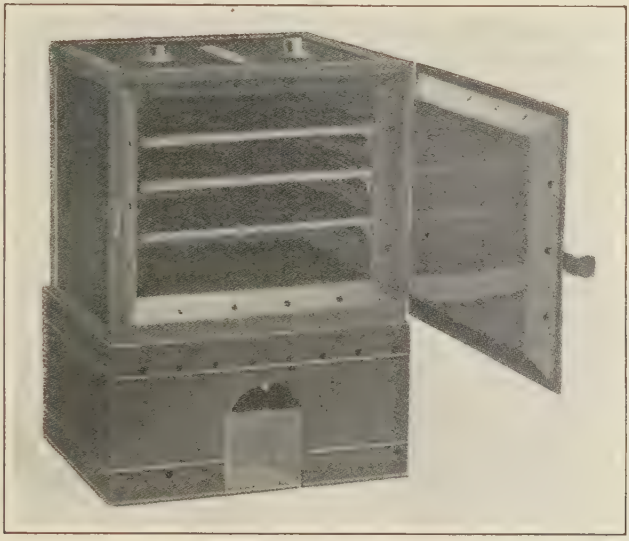


Fig. 143.—Hot Air Sterilizer.

The ordinary household cooking oven is as good as any specially contrived apparatus for the disinfection of small objects by dry heat. In the absence of a thermometer it is usual to heat the oven to a point necessary to brown cotton and expose the objects no less than one hour.

Boiling.—Boiling is such a commonplace, every-day procedure that it is often neglected in practical disinfection despite the fact that it is one of the readiest and most effective methods of destroying infections of all kinds. An exposure to boiling water at 100° C. continued for an hour will destroy the living principles of practically all the infectious diseases with which we have to deal in public health work. To be sure, there are a few spores that have shown a remarkable resistance to boiling water and streaming steam in laboratory experiments. Boiling, therefore, cannot be entirely depended upon where tetanus, anthrax, or resisting spores are in question. As a matter of fact, a degree of moist heat much lower than the boiling point of water is effective against the great majority of the known viruses. Thus a temperature of 60° C. for 20 minutes will destroy the microorganisms of cholera, typhoid, dysentery, diphtheria, plague, tuberculosis, pneumonia, erysipelas, and practically all non-spore-bearing bacteria. Boiling kills them at once.

Boiling is especially applicable for the disinfection of bedding, body linen, towels, and fabrics of many kinds; also kitchen and tableware, cuspidors, urinals, and a great variety of objects. Surfaces, such as floors, walls, beds, metal work, etc., may be effectively disinfected by mechanically cleansing them with boiling water. The efficacy of boiling water, especially when used in such circumstances, is greatly increased by the addition of corrosive sublimate, carbolic acid, or one of the alkaline coal-tar creosotes. The addition of lye, borax, or a strong alkaline soap also increases the penetrating and detergent power of boiling water when applied to surfaces soiled with organic or oily matters.

In using boiling water for the disinfection of bright steel objects or cutting instruments the addition of 1 per cent. of an alkaline substance such as carbonate of soda will prevent rusting and injury to the cutting edge.

Steam.—Steam is one of the most satisfactory disinfecting agents we possess. It is reliable, quick, and may be depended upon to penetrate deeply. Further, it does more than disinfect; it sterilizes. Vegetating bacteria are killed instantly and most spores in a few minutes. It may therefore be used to destroy the infection of any one of the communicable diseases.

Either streaming steam or steam under pressure is used in practical disinfection.

Streaming steam has the same disinfecting power as boiling water, and an exposure of half an hour to an hour is sufficient. Steam under pressure is a more powerful germicide than streaming steam. At a pressure of 15 pounds to the square inch steam has a temperature of approximately 120° C. and may be depended upon to sterilize in 20 minutes. At 20 pounds pressure it has a temperature of approximately 125° C. and will sterilize in 15 minutes.

Steam is applicable to the disinfection of bedding, clothing, fabrics of all kinds, and a great variety of other objects, provided certain precautions are taken to prevent shrinking, staining, running of colors, etc. Steam shrinks woolens and injures silk fabrics; it ruins leather, fur, skins of all kinds, rubber shoes, oilcloth, and articles made of impure rubber or containing glue, varnish, or wood.

It is important in disinfecting with steam, whether with streaming steam or steam under pressure, to expel the air from the apparatus. The air, being a poor conductor of heat, forms dead spaces and prevents the steam's coming in direct contact with the articles to be disinfected, thereby defeating the object to be attained. As steam is lighter than air the latter can best be expelled from the apparatus by admitting the steam from above, in which case the descending column of steam forces the air out at the bottom. If the steam is admitted at

the bottom it swirls up, making a nearly uniform mixture with the air, and while the temperature quickly rises in the apparatus the air escapes mixed with the steam, so that it takes a long time and an unnecessary waste of steam to drive out the contained air.

Disinfection with streaming steam may be accomplished in many ways without the use of special apparatus. For rough and ready work on the railroad the objects to be disinfected may be hung in a freight-car and the steam brought from the locomotive. On board a vessel one of the compartments above the water-line may be filled with steam from the boiler. Objects may be steamed in any rough structure wherever a boiler is found to furnish the steam. Such a structure need not be tight, for the streaming steam escaping from the cracks produces a circulation and favors penetration.

In the laboratory small objects are disinfected in streaming steam in the Arnold steam sterilizer or the Koch steamer.

On account of the great certainty with which steam under pressure acts it is the favorite method in practical disinfection, especially where sterilization is required, and devices for applying this process on a large scale have reached a high degree of perfection. The smaller forms of steam sterilizers under pressure are known as digestors or autoclaves and the larger ones as steam disinfecting chambers.

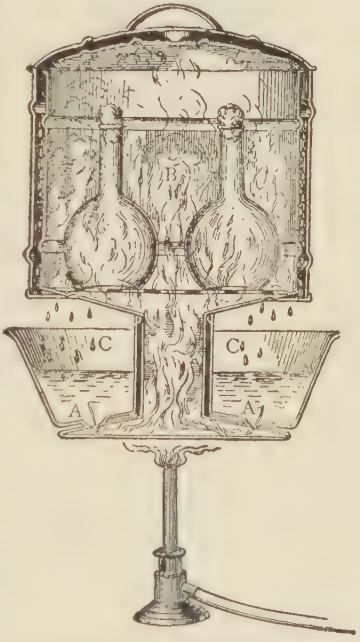


Fig. 144.—Section through Arnold Steam Sterilizer.

The Autoclave.—The autoclave, digestor, or steam sterilizer consists of a closed kettle usually made of copper and sufficiently strong to withstand the pressure. Water is placed in the kettle and the heat is applied to the bottom, usually by means of several Bunsen gas jets. The apparatus is surrounded as high as the shoulder, where the lid is attached, with a metal jacket which serves the purpose of bringing the heat of the flame in contact with the entire surface of the kettle. The lid is made to fit tightly by means of screw bolts and a rubber gasket. A thermometer, pressure gage, safety valve and a small opening with a stop-cock for the purpose of allowing the escape of the air are provided. If all the air is not expelled from the apparatus the dead spaces will have a much lower temperature than that registered on the thermometer. For instance, the steam itself may register a tem-

perature of 130° C., while test fluids exposed may only reach 70° to 80° C. Therefore, in using this form of sterilizer it is customary to allow the steam to escape in full force for several minutes before permitting the pressure to rise.

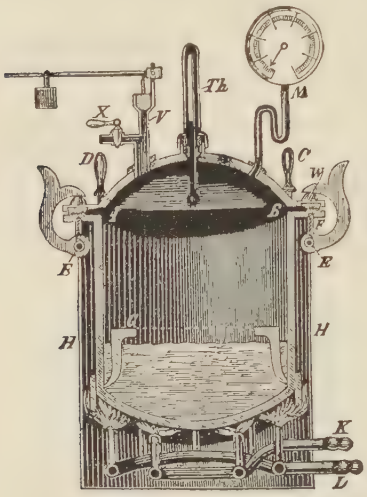


Fig. 145.—Section through Autoclave.

In the sterilization of liquids, for which this apparatus is frequently used, it is important, at the conclusion of the process, not to take off the lid or open the valves, or in any other way release the pressure until the apparatus has cooled; otherwise the condensed steam causes a diminished pressure, in which the heated liquids will boil energetically, resulting in a bubbling over, a blowing out of stoppers, or a bursting of the flasks. It is therefore necessary to wait until the pressure is zero, as registered on the gage; or, better, until the condensing steam produces a partial vacuum and the air is automatically sucked in through the vacuum valve, which is sometimes fitted in the lid of the apparatus for this very purpose.

The Steam Chamber.—The steam disinfecting chamber has reached a high degree of usefulness through the gradual perfection of the details of its working parts. These chambers are somewhat complicated and their mechanical construction must be mastered in order to insure reliable results. Steam disinfecting chambers may be used with streaming steam or with steam under pressure; with formaldehyd gas alone, or with this gas in combination with dry heat; and, finally, with various combinations of these methods with or without a vacuum.

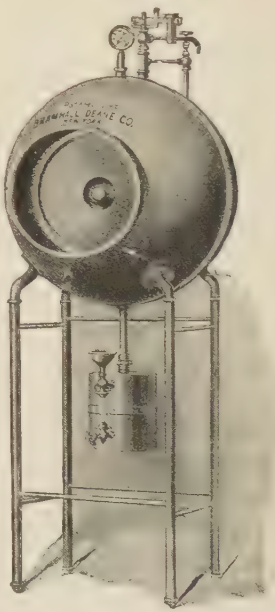


Fig. 146.—Bramwell-Deane Steam Sterilizer.

The disinfecting chamber itself may be rectangular or cylindrical in shape, the former giving more effective space, the latter being a stronger and cheaper method of construction. The chamber is built of an inner and outer shell forming a steam jacket, as shown in Fig. 147. The steam jacket serves several purposes. By heating the contents of the dis-

infecting cylinder before the steam is turned in it avoids condensation. During the process of disinfection it helps keep the steam in the chamber "live," thereby preventing the wetting of the objects exposed. After the disinfection is finished and the chamber opened the heat from the steam in the jacket may be used to dry the objects which have just been steamed. Therefore, in using this apparatus for disinfecting with steam, either with or without pressure, the steam is kept circulating in the jacket from the beginning to the end of the process.

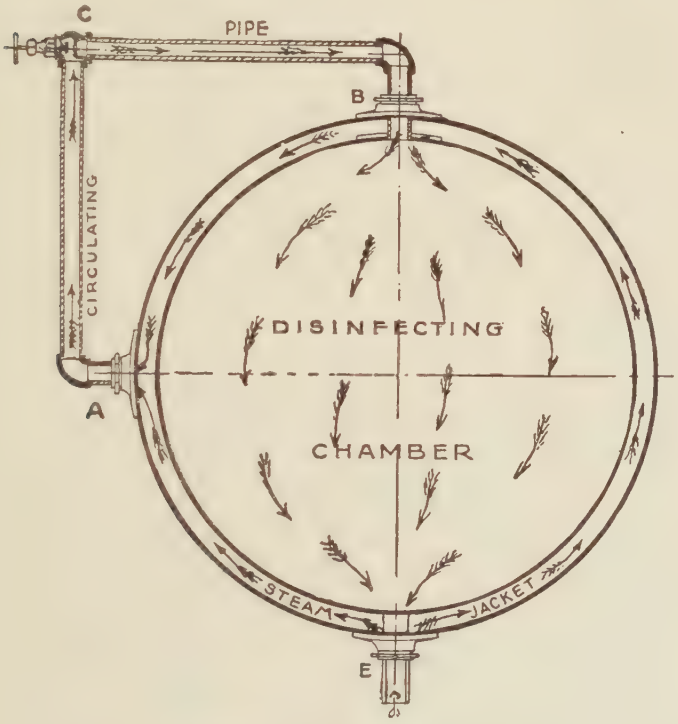


Fig. 147.—Cross Section through Steam Disinfecting Chamber.

In the jacket the steam has a perfectly free circulation, so that the entire disinfecting cylinder, with the exception of the doors, is surrounded by live steam. The outer shell of the jacket is insulated with a covering of sectional magnesia, asbestos, or some other non-conducting substance.

The steam from the boiler passes through the main steam pipe A (Fig. 149) to the pressure-reducing valve (2), and thence to the bottom of the jacket at B, B.

Into the disinfecting chamber itself the steam can be admitted only from the jacket, through the circulating pipes, A, C, B (Fig. 147, and after circulating through the disinfecting chamber in the

direction as shown by the arrows is allowed to pass out with the drip through the drain D (Fig. 148). Upon the completion of the process the steam may be blown off through the vacuum pipe F,

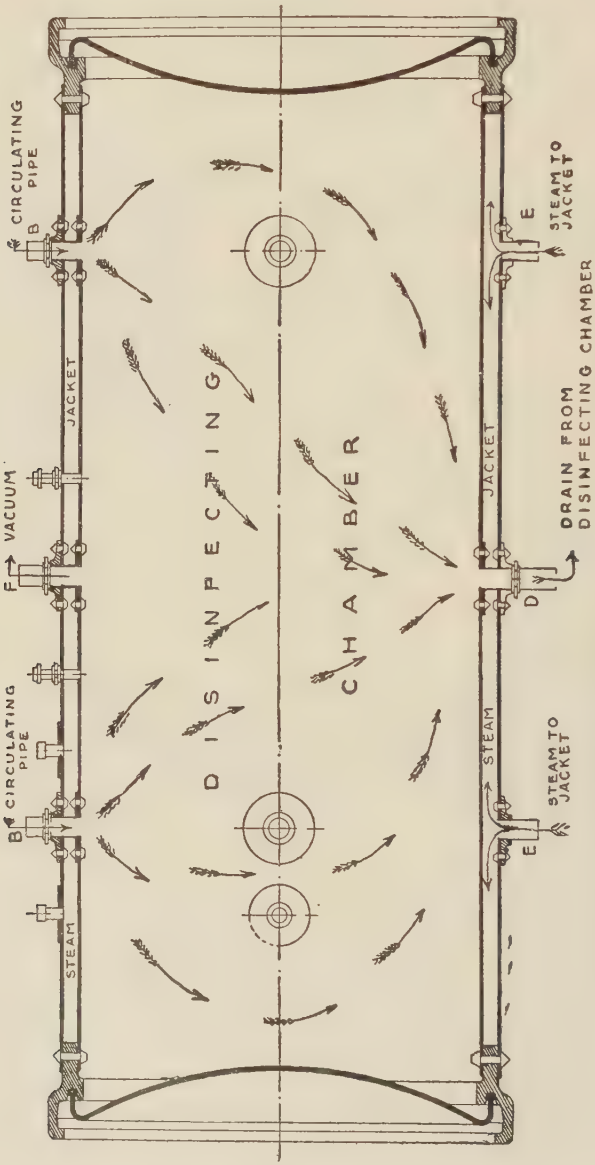


Fig. 148.—Longitudinal Section through Steam Disinfecting Chamber.

but this outlet should not be used during the steaming because the desired circulation would not be obtained.

It will be noticed that the steam is admitted to the bottom of the jacket, but to the top of the disinfecting chamber, which is designed

to favor the expulsion of the air through its outlet at the bottom by means of the descending column of steam. Therefore, in order to expel all the air and fill the chamber with steam it is essential to open the drain D (Fig. 148) while the steam is entering through B, B, and this outlet, D, should not be closed until steam escapes freely. In using the vacuum attachment to expel the air contained in the apparatus the *modus operandi* is somewhat different.

A partial vacuum may be obtained in steam chambers of this type with the ejector (4, Fig. 149). The object of the vacuum is to facilitate the penetration of the steam, which rushes into all the interstices of fabrics and inaccessible places, to take the place of the air which has been withdrawn. The ejector works upon the familiar principle of the water vacuum pump, the air being drawn or sucked along with the current. With a pressure of 80 pounds in the boiler and the valve J (Fig. 149) wide open the ejector will produce a partial vacuum of 15 inches in one of the largest sized chambers in one minute, which is very much quicker than can be accomplished with the ordinary forms of piston pumps.

Any steam disinfecting chamber may have attached to it an apparatus for generating formaldehyd gas, so that objects that are injured by exposure to steam may be disinfected with formaldehyd, plus dry heat. Before the formaldehyd is admitted into the chamber a partial vacuum may be established by means of the ejector. In this way the penetration of the gas is very much facilitated.

In the best patterns the steam disinfecting cylinders are open at both ends, so that the infected objects may be introduced from one side and taken out from the other, which diminishes the risk of reinfecting them. The joint between the door and the chamber is made tight by means of a heavy rubber gasket. The door should not be pressed against this gasket more firmly than is found necessary to retain the steam, otherwise the rubber will soon be rendered useless. When not in use the door should be kept open, otherwise on cooling the metal will adhere to the rubber gasket. This may be prevented to a certain extent by keeping the rubber gasket covered with graphite.

In addition to the above-mentioned attachments the disinfecting chambers are also supplied with a thermometer (7, Fig. 149), the stem of which is turned at right angles and protrudes so as to indicate the temperature of the interior of the disinfecting chamber. The thermometer, however, is so close to the jacket that it is influenced by the heat in the jacket, which is usually higher than the temperature of the interior of the chamber. The thermometer should be in the door, or differently arranged, to give trustworthy results. In disinfecting with steam under pressure the pressure, as indicated by the gage, is a more reliable guide than the temperature registered by the thermometer.

There are forms of mercurial and metallic thermometers that make an electric contact when a certain temperature is reached, and which may be connected to ring a bell. They have a decided advantage in that they may be placed anywhere within the chamber, even in the center

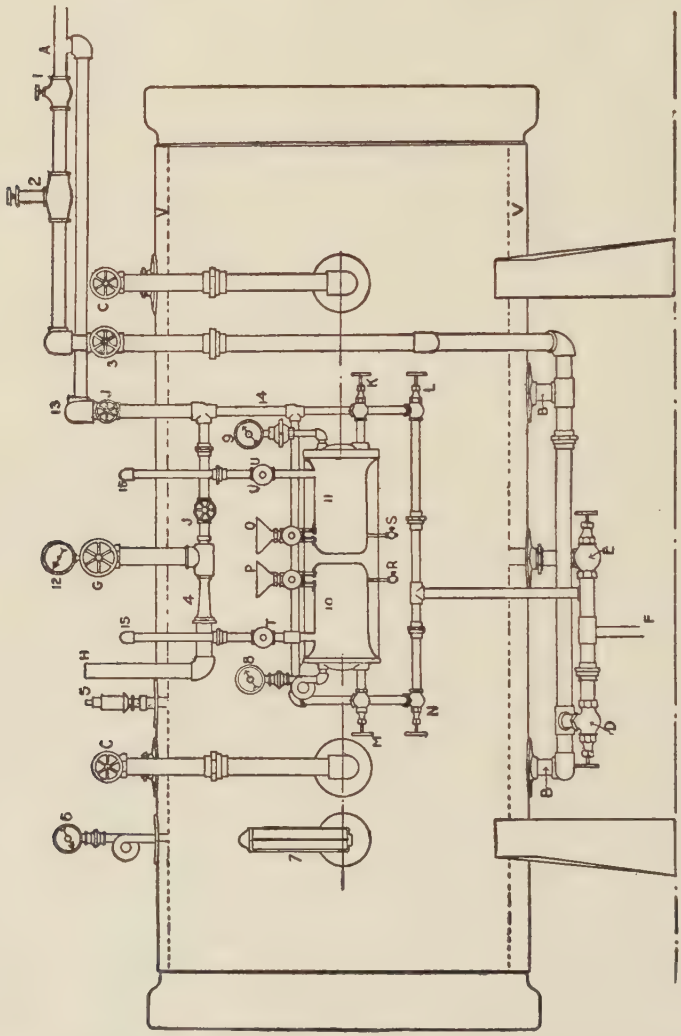


Fig. 149.—Kinyoun-Francis Steam Disinfecting Chamber.

of bundles, etc., and are more trustworthy than any form of mercurial instrument fastened through the heavy metallic walls of the apparatus.

An ingenious form of thermometer, made to register when the temperature reaches 100° C., has been designed by Merk, and is shown in the accompanying illustration (Fig. 150). A small stick of the metallic substance which is supplied with the instrument and which

melts at exactly 100° C., fastened at A, keeps the electrodes at B and C apart. The entire thermometer D is then placed in the box E for protection, and this is placed in the chamber or in the inside of bundles to be disinfected. The insulated wires from F and G are connected with a battery and bell. As soon as the temperature reaches 100° C. the little metal stick melts, the contact is made between B and C, and the bell rings. This form of thermometer is more accurate than the pyrometers, which depend upon the contact being made by the unequal expansion of a compound metal bar, for the reason that moisture collects upon the electrodes and an electric contact is sometimes made before the metal parts actually touch, thereby giving false readings.

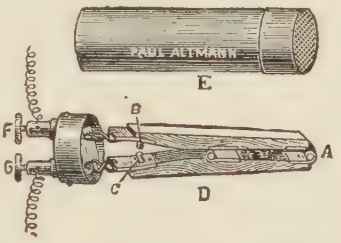


FIG. 150.—AUTOMATIC THERMOMETER.

Steam chambers must always be provided with galvanized or copper hoods to prevent rust-stained drip from soiling the clothing and other objects exposed to the steam; gages to indicate both vacuum and steam pressure, and a safety valve to prevent over-pressure in the chamber. The amount of pressure from the boiler is regulated by a reducing valve in the main steam pipe.

For convenience in handling the goods cars are provided, of light wrought-iron construction, with movable trays made of galvanized screens; also bronze hooks at the top of the car, permitting the articles to be laid upon the trays or to be hung up on the hooks.

In the accompanying diagram (Fig. 151) the method of installing the steam chambers in the disinfecting shed of a quarantine station is shown. It will be noted that the cylinders open on both ends, and that a dividing wall running across the building separates the receiving end, where the infected objects arrive and are prepared for disinfection, from the discharging end, where the contents of the chamber are aired, dried, and repacked after disinfection.

This separation is essential where a large amount of disinfection is done for a variety of diseases, as, for example, in a municipal disinfecting establishment or at the quarantine station of a busy port. It is true that the infection of certain diseases is not apt to contaminate the surroundings, and in such cases there would be little risk in taking the disinfected articles out of the same end of the chamber from which they are put in, especially if the exposed surfaces are mopped with a disinfectant in the interim. But this is a risk that need not be taken; in fact, all well-regulated disinfecting plants maintain a rigid separation between the two sides, never allowing both doors of the chamber to be open at the same time, and providing two sets

of workmen, one for the "infected" and one for the "disinfected" side.

The chambers must be loaded with care in order to obtain reliable results and to avoid injuring the articles exposed to the process. The packages must not be too large or crowded too closely, for, although the vacuum facilitates the penetration of the steam, there is a limit in this regard; it takes so much longer for disinfecting agents to penetrate dense packages and bundles that there is little saving of time and a distinct loss in trustworthiness. Steam cannot penetrate compressed bundles of rags, bales of cotton, feathers, hair, or other packages of merchandise which are often presented for disinfection. Fortunately, it is seldom necessary to disinfect such packages. When, however, this is called for it is essential to open and properly expose such objects to the action of the disinfecting agent.

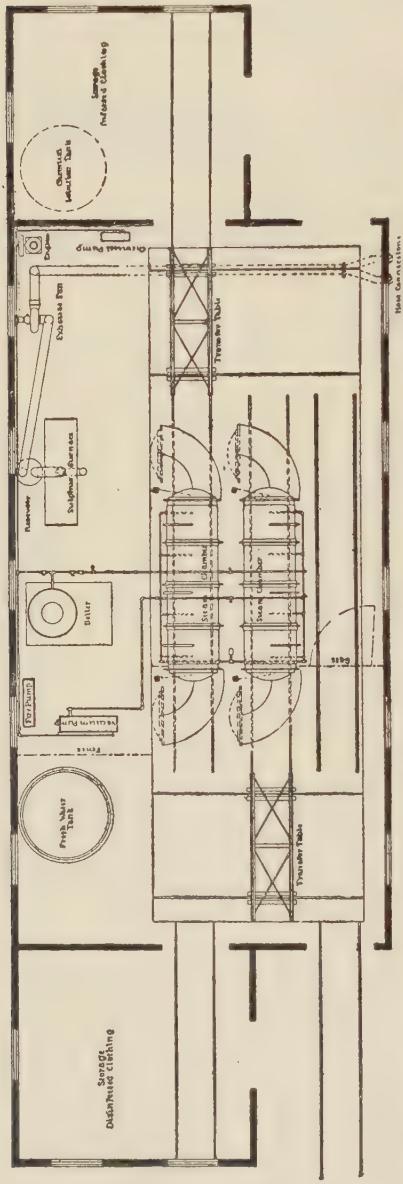


FIG. 151.—PLAN SHOWING THE METHOD OF INSTALLING THE DOUBLE-ENDED STEAM CHAMBERS AT A NATIONAL QUARANTINE STATION.

In the municipal disinfecting stations of Paris the process of applying steam under pressure is as follows: The pressure is brought up to 15 pounds in the chamber and held there five minutes; then released. The pressure is again brought up to 15 pounds, held there five minutes, and again released. This is repeated three times, when the disinfection is completed. The cylinders are fitted with an ingenious arrangement for the automatic registration of the process. Each chamber is connected by a small copper tube to a register with a moving pen and revolving drum carrying a chart. The horizontal lines 1 to 10 on the chart each represent a pressure of one-tenth of an atmos-

phere, and the vertical lines represent five minutes in the revolution of the drum. Each steaming is represented thus:

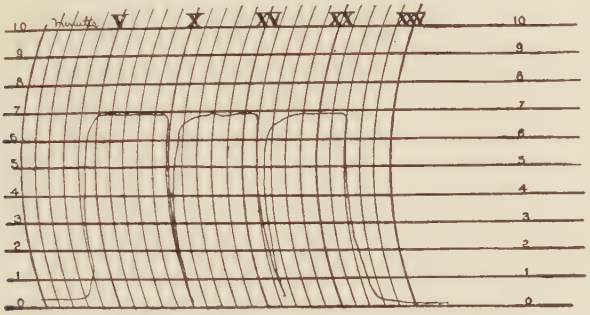


Fig. 152.

These charts, which can be removed only by the chief of the station, are sent each day to the Inspector-General, and give a perfect guarantee that each steaming has been done as directed.

CHAPTER III

CHEMICAL AGENTS OF DISINFECTION

GASEOUS DISINFECTANTS

A gas is an ideal weapon for destroying such invisible foes as we have to deal with in public health work, especially for terminal disinfection. By reaching all portions of a room or confined space a gas lessens the risk of overlooking any surface upon which the infective agent may be lodged, but the ideal gas for this purpose is still to be discovered.

There is practically only one gas suitable for general application, viz., formaldehyd. This substance comes nearer being an ideal disinfectant than any of the gases in general use. It is not poisonous, does not injure fabrics, colors, metals, or objects of art and value. Formaldehyd, however, has distinct limitations, which are dealt with more in detail under the description of the gas.

Sulphur dioxid is too destructive for fabrics, colors, and metals for general use. It is a better insecticide than germicide. It is very poisonous to all forms of animal life, which makes it valuable in disinfection against insect and animal-borne diseases. It has no equal for the fumigation of the holds of ships, cellars, sewers, stables, and other rough structures infested with vermin.

The very poisonous and destructive nature of chlorin gas contracts its usefulness to narrow limits.

Hydrocyanic acid gas is too poisonous to use in the household, and is limited in practice to the destruction of infection and vermin on board ships, in warehouses, greenhouses, granaries, railroad cars, and other uninhabited or isolated structures.

None of the gaseous agents can be depended upon for more than a surface disinfection. They all lack the power of penetration.

Preparation of the Room.—The preparation of a room or space to be disinfected with a gas is a matter of some importance. A larger amount of gas than is thought possible is lost through leaks by diffusion and absorption and in other ways; therefore the room should be made tight, all cracks and crevices should be well closed by pasting paper over them or by caulking with suitable material of some kind. Do not

forget to close the registers, flues, hearths, and ventilators, and look carefully for openings in out-of-the-way places. Then expose the objects in the room so that the gas may have ready access to all the surfaces. Hang clothing, bedding, and fabrics upon lines strung across the room; move bureaus, beds, and furniture away from the walls, open doors of closets, drawers of bureaus, lids of boxes, and the like so that the gas may freely enter and diffuse to all corners.

While the articles in the room must be arranged so that the gas may freely gain access to all surfaces possible, the mistake must not be made of going to the opposite extreme of disarranging the contents of the room too much, for the same surfaces should be exposed to the gas that were exposed to the infection.

The strength of the gas and time of the exposure necessary to insure disinfection have been determined by exact laboratory experiments, but the conditions found in actual practice are so variable that we must allow for a liberal excess to make up for inevitable wastage. Wind pressure also seriously influences the efficiency of gaseous disinfectants in a confined space. Much more air than is commonly thought possible forces its way through cracks and through the walls themselves. The wind pressure may thus drive the fumigating gas entirely away from one side of the room. It is only necessary to stand upon the leeward side of a structure being disinfected with sulphur dioxid or formaldehyd to realize the great quantity of gas blown from the enclosure.

Formaldehyd Gas.—Formaldehyd [1] is the most generally useful and one of the best disinfecting gases that we possess. Its superiority depends upon its high value as a germicide, its non-poisonous nature, and upon the fact that it is not destructive. The secret of successful disinfection with this substance is to obtain a large volume of the gas in a short time.

Formaldehyd (HCHO) exists in at least three well-recognized isomeric states:

(1) Formaldehyd (formic aldehyd) is a gas at ordinary temperatures, colorless, and possessing slight odor, but having an extremely irritating effect upon the mucous membranes. At a temperature of about —20° C. the gas polymerizes into paraformaldehyd, known commercially as paraform.

(2) Paraform is a white substance, unctuous to the touch, soluble in both water and alcohol. It consists chemically of two molecules of formaldehyd. It is this substance which is supposed to compose the commercial solutions of formaldehyd known as formalin, formol, etc.

(3) Trioxymethylene is formed by the union of three molecules of

[1] Formaldehyd is the gas, formalin is the aqueous solution of the gas.

formaldehyd. It is a white powder giving off a strong odor of the gas. It is but slightly soluble in alcohol and water.

Formaldehyd gas possesses about the same specific gravity as air; it diffuses slowly, although somewhat better than sulphur dioxid. Formaldehyd combines with nitrogenous organic matter. A few drops added to the white of an egg will prevent its coagulation by heat. The formaldehyd unites with the albumin to form a totally new compound. Combined with gelatin it keeps that substance from liquefying. It is from this property of combining directly with the albumins forming the protoplasm of the microorganisms that formaldehyd is supposed to derive its power as a germicide. It is perfectly plain, therefore, why there must be direct contact between the gas and the germ in order to accomplish disinfection.

Formaldehyd also unites readily with the nitrogenous products of fermentation and decomposition, forming new chemical compounds which are both odorless and sterile. It is thus a true deodorizer in that it does not mask one odor by another still more powerful, but forms new chemical bodies which possess no odor.

Formaldehyd apparently has no detrimental effects upon silks, woolens, cotton, and linen. It does not change colors, with the exception possibly of a slight effect upon some of the delicate anilin lavenders. An oil painting is not perceptibly altered after prolonged exposure to the gas. The metals are not attacked. It is this non-destructive property of the gas that renders it generally applicable. It is practically the only gaseous germicide which can be used in the richest apartments, containing objects of art and value, without fear of damage.

The commercial solutions known as formalin are said to contain 40 per cent. of formaldehyd gas. They are not always up to standard (average 36 per cent.), and, being volatile, there is a certain loss if not well kept. In winter there is a decided deterioration, owing to the polymerization and precipitation of trioxymethylene. This substance is often found in abundance at the bottom of the bottle or carboy as a white precipitate. For these reasons it is well to use an excess of the liquid in practical work if the exact strength of the formalin has not recently been determined.

Formalin solutions of commerce are almost all acid in reaction, due in part to formic acid. Some of the commercial solutions also contain a certain amount of wood alcohol (about 10 per cent.) which is added to increase their solubility and stability.

A certain amount of heat and moisture is necessary to obtain successful disinfection with formaldehyd gas. The exact amount of moisture necessary depends somewhat upon the temperature. As a general working rule it may be stated that if the temperature is below

65° F. or if the relative humidity is below 60 per cent. the results become irregular; much below these figures the results are unreliable. Formaldehyd polymerizes at low temperatures, therefore in cold weather it may be necessary to artificially warm the room to be disinfected. In dry weather moisture should be added to the room.

Formaldehyd gas cannot be depended upon to accomplish more than a surface disinfection. Under ordinary circumstances it possesses small powers of penetration. It takes a large volume and a long exposure to penetrate several layers of thin fabrics. The gas polymerizes in the meshes of the fabric and is deposited as paraform upon surfaces. Large quantities of the formaldehyd are lost by uniting chemically with the organic matter of fabrics, especially woolens, which further hinders its penetration. Therefore, formaldehyd gas cannot be relied upon to disinfect fabrics, especially quilted goods and materials requiring deep penetration.

Formaldehyd gas has the power of killing spores, although not with sufficient certainty to render it a trustworthy agent for infections such as anthrax and tetanus. It has the great advantage of killing dry microorganisms, although not quite so readily as when they are moist.

Bacteria exposed directly to the action of a concentrated volume of formaldehyd gas are killed almost instantly. Under similar conditions spores are killed within an hour, but in practical work it is necessary to prolong the time of exposure, as it takes considerable time for it to permeate to all the corners and dead spaces of a room. Bacteria are not always directly exposed upon the surface of objects, as they are in laboratory experiments, and, furthermore, they are frequently imbedded in albuminous matter or in dust, both of which retard the action of the gas.

Formaldehyd gas is not toxic to the higher forms of animal life, although it stands at the head of the list of germicides. Long exposure to weak atmospheres of the gas sufficient to kill germs has but slight effect upon animals. Guinea-pigs, rats, mice, and rabbits exposed to concentrated atmospheres obtainable by any of the methods for evolving it are not killed after half an hour's exposure. The only effect produced is a violent irritation of the mucous membranes of the respiratory tract, from which the animals may subsequently die. Microorganisms exposed to this same concentration of the gas are killed almost instantly.

Formaldehyd is not an insecticide. In the strongest volumes of the gas obtainable it seems to have practically no effect upon roaches, bedbugs, and insects having strong chitinous coverings. It may kill the frailer insects, but its action is uncertain; thus mosquitoes may live in a weak atmosphere of the gas over night.

Upon the completion of the time required to disinfect a room it is best to open all the doors and windows and let the gas blow away.

This may be a troublesome procedure. If the windows can be reached from the outside it is easy enough, but if the room must be entered it is advisable for the operator to cover his mouth and nose with a moist towel and act quickly. It was formerly the custom to neutralize the gas with ammonia, but this is little practiced now. The ammonia neutralizes the formaldehyd by the production of hexamethylene-tetramine.

The following methods are used for disinfection with formaldehyd gas:

(1) Autoclave under pressure.
(2) Retort without pressure.
(3) Generator or lamp.
(4) Formaldehyd and dry heat in partial vacuum.
(5) Spraying.
(6) Heating paraform.
(7) Potassium permanganate and formalin.
(8) Formalin, lime, and aluminium sulphate.

The most generally useful of these methods are the last two. They have the great advantage of simplicity, of dispensing with all apparatus, and of evolving a large amount of the gas in a short time.

THE PERMANGANATE-FORMALIN METHOD.—Use 500 c. c. of formalin and 250 grams of potassium permanganate for each thousand cubic feet of air space. The permanganate is first placed in a bucket or basin and the formalin poured upon it. An active effervescence takes place and considerable heat is evolved; therefore a pail of sufficient capacity, and especially of sufficient height, should be used to prevent splashing or boiling over. In Board of Health work it is advisable to have galvanized iron pails made for this purpose with a flaring top. The floor should be protected against the heat by placing the bucket upon a brick, board, or other suitable device.

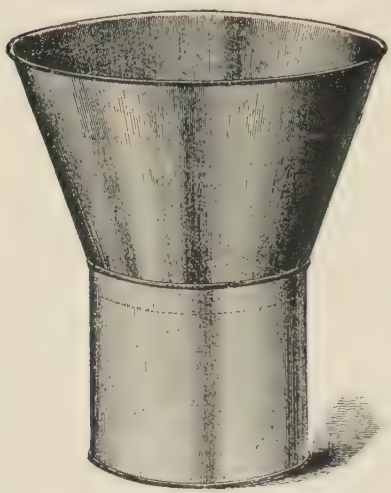


FIG. 153.—FLARING TOP TIN BUCKET FOR GENERATING FORMALDEHYD BY THE PERMANGANATE METHOD. Height 15 inches, diameter 10 inches at base, 15 inches at top of flare.

When the permanganate of potassium and formalin are brought in contact very active oxidation takes place, with the production of formic acid and heat. It is the heat that liberates the formaldehyd gas. Chemically, therefore, the method is a wasteful one, but practically a very serviceable one. It was first de-

scribed by Johnson of Sioux City, Iowa, in 1904. In the same year Evans and Russell of Augusta, Maine, used the method.

The Formalin-Lime and Aluminium-Sulphate Method.—This method was first described by Walker of the Department of Health, Brooklyn, N. Y. It is somewhat slower than the potassium permanganate method, but otherwise appears to be just as efficient.

The proportions for each 1,000 cubic feet are as follows:

Sol. A.—Aluminium sulphate..... 150 grams
Dissolved in hot water..... 300 c. c.
Sol. B.—Formalin (40 per cent. CHOH)... 600 c. c.
Lime.—Unslaked lime .....2,000 grams
Mix solutions A and B and pour upon the lime.

In practical work 20 to 25 pounds of the commercial aluminium sulphate is dissolved in 5 gallons of hot water. This is sufficient to mix with 15 gallons of a 40 per cent. formaldehyd solution and then used in the proportions as stated above. The lime should be freshly burned, broken into small particles, and should slake rapidly in cold water. The lime is placed in a large bucket. The formalin and aluminium sulphate solutions should be mixed and poured over the lime. In a few minutes the lime begins to slake and the heat evolved drives off the formaldehyd gas.

The Spraying Method.—Spraying formalin is a satisfactory and simple method of disinfecting small inclosures, such as wardrobes, closets, and cabinets. It is not practical for larger rooms. If the formalin is sprayed directly upon the objects to be disinfected they enjoy the direct germicidal action of the substance in solution and, further, are bathed in the gas which is slowly evolved. The method is particularly serviceable for the disinfection of bureau drawers, closets, and small spaces. When used to disinfect small rooms suspend a bed sheet from a line stretched across the middle of the room. An ordinary bed sheet presenting a surface of about 2 by 2½ yards is required for every 1,000 cubic feet of space of the room. Properly sprinkled this will carry, without dripping, 8 ounces of formalin. The ordinary sprinkling pot used by florists can be used to spray the sheets. The room should remain closed not less than 8 hours.

The other methods for disinfecting with formaldehyd gas are not described because some of them are unreliable, and none of them are as serviceable in practical work as the formalin-permanganate method or the formalin-lime method.

Sulphur Dioxid.—Sulphur dioxid (SO_2) is not a very efficient germicide, but is exceedingly poisonous to mammalian and insect life. It is this property which makes it of especial value as a fumigant against

diseases spread by rats, mice, flies, fleas, mosquitoes, etc. For this purpose it has no superior.

The action of sulphur dioxid as a germicide depends upon the presence of moisture. The dry gas is practically inert against bacteria. Sulphur dioxid cannot be depended upon where penetration is required. Its action is merely upon the surface. It does not kill spores.

Sulphur dioxid possesses the advantage of being cheap and readily procurable. There is hardly a crossroad store in the country where a reasonable quantity of sulphur, either in the form of flowers or in rolls or sticks under the name of brimstone, cannot be obtained. Sulphur dioxid is especially applicable to the holds of ships, freight-cars, granaries, stables, out-houses, and similar rough structures—particularly if infested with vermin.

The disadvantages of sulphur dioxid as a disinfecting agent are such as to contract its application to rather narrow limits. It bleaches all coloring matter of vegetable origin and many anilin dyes. It attacks almost all metals; it acts upon cotton and linen fabrics so as to seriously weaken their tensile strength, especially if starched.

Sulphur dioxid is a heavy, colorless, irrespirable gas with a peculiar suffocating odor and irritating properties. It has a density of 2.4. On account of the heavy specific gravity as compared to air it diffuses slowly and then settles toward the bottom of the compartment.

Cold water takes up more than 30 times its volume of sulphur dioxid. The solution contains sulphurous acid (H_2SO_3), and it is in reality this acid that is the disinfecting agent. The dry gas is therefore inert and moisture is essential in order to obtain any germicidal effect. It is also this acid and some sulphuric acid which has such a destructive effect upon fibers, colors, and metals. The corrosive action of these acids upon fabrics takes place slowly, and the damage may largely be obviated if they are washed at once. Metal work may be protected by coating it with a thin layer of vaselin or heavy-bodied oil.

Sulphur dioxid may readily be condensed into a clear liquid by either cold or pressure or a combination of both. At ordinary atmospheric pressure it condenses if the temperature is reduced to — 18° C., which is about the temperature of a mixture of ice and salt. At ordinary temperatures it liquefies if the pressure is raised to about four atmospheres, that is, 60 pounds. This liquid is a stable substance when kept well sealed and protected from the action of the air. It rapidly volatilizes when poured into an open vessel. It is now found in commerce and is one of the methods used for producing the gas for fumigating purposes.

The complete combustion of 1 pound of the sulphur in a space 1,000 cubic feet will produce 1.115 per cent. of sulphur dioxid, but this amount cannot be obtained in practice because the sulphur of com-

merce contains impurities such as sulphate of lime and sand, and a portion of the burning sulphur is always oxidized to the formation of ill-defined compounds. Therefore one pound may be considered as producing approximately 1 per cent. of the gas by being burned in 1,000 cubic feet of space, and five pounds will generate approximately 5 per cent., which is the maximum theoretical amount obtainable by burning sulphur in a confined space. This is the amount usually used when a germicidal action is desired.

The amount of moisture necessary to convert sulphur dioxid into sulphurous acid is readily computed. It will be found that one-fifth of one pound of water should be volatilized or added for each pound of sulphur burned. The water may be added in the form of steam or in the form of a finely divided spray, or it may be vaporized by the heat generated by the combustion of the sulphur itself. The latter method is the one that commends itself in practical use, and is described under the "pot method."

While moisture is essential for the germicidal action of sulphur dioxid, it is not necessary in order to kill insects and the higher forms of life. Dry sulphur dioxid is quite as efficacious against rats, mice, fleas, flies, mosquitoes, bedbugs, roaches, etc., as the moist gas.

In disinfecting with sulphur dioxid it is necessary to tightly seal the compartment. The gas is disengaged so slowly that it may escape through cracks and crevices almost as fast as it is formed. In cold weather the heating of the room to be disinfected will greatly aid in the disinfecting action of the gas.

There are three well-recognized methods of fumigating with sulphur dioxid, viz., (1) the pot method, (2) liquid sulphur dioxid, (3) sulphur furnace.

The Pot Method.—The pot method is at once the easiest, cheapest, and probably most efficient method of using sulphur dioxid. The only materials required are iron pots and some sulphur. The best way to apply the method is by placing the sulphur in large, flat, iron pots known as Dutch ovens. Not more than 30 pounds of sulphur should be placed in each pot. The sulphur is preferably used in the form of flowers of sulphur. If it is in sticks or rolls it should be crushed into a powder, which may conveniently be done by placing the sulphur in a stout box and pounding the lumps with a heavy timber. The pot holding the sulphur should be placed in a tub of water, as shown in Fig. 154. The water not only diminishes the danger from

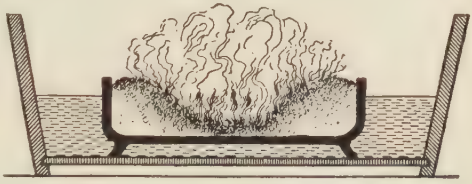


Fig. 154.—The Pot Method of Burning Sulphur.

fire and protects the floor, but by its evaporation furnishes the moisture necessary to hydrate the sulphur dioxid, upon which the disinfecting power of the gas depends. Thus the moisture is furnished automatically and does away with the necessity for its introduction by means of steam or a spray. Although the specific gravity of sulphur dioxid is greater than that of air, when hot it rises, aided by the upward current produced by the burning sulphur. Hence the pots should not be on the floor or at the bottom of the hold in the case of vessels, lest the cold gas settle and the flame, being deprived of oxygen, be extinguished before all the sulphur is burned. The pots may therefore be placed upon a table or box or, in the holds of ships, upon piles of ballast or on the "'tween decks."

Roberts and McDermott[1] suggest that the sulphur be burned upon pans arranged upon a rack as shown in Fig. 155, instead of pots. The

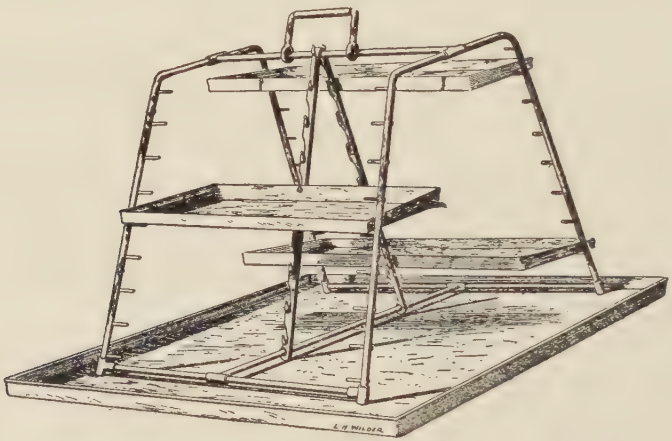


FIG. 155.—LARGE STACK BURNER FOR SULPHUR, WITH 15 OF THE 18 PANS REMOVED TO SHOW CONSTRUCTION.

advantages of this stack burner are that a large amount of sulphur may be more quickly burned in less time than is possible with the pot method. Further, the intense heat below each pan in the stack burner aids the complete and rapid burning of sulphur in the pans above it. A stack burner will burn sulphur of too poor a quality to give any satisfaction in the pots. The ground sulphur is placed in the pans, the surface of the sulphur is moistened with alcohol, and ignited. Each shelf should be lighted separately to save time. The upper pan or pans may be filled with water to hydrate the sulphur dioxid necessary for its germicidal action.

[1] *Public Health Reports*, U. S. P. H. and M. H. S., March 31, 1911, Vol. XXVI, 13, p. 403.

The sulphur may be lighted by means of hot coals or a wood fire, but the most reliable way to get it well lighted is by alcohol, turpentine, or kerosene on a pledget of waste. Make a little crater of the sulphur, soak liberally with alcohol, and ignite. The sulphur then burns in the center, and as it melts runs down from the sides and forms a little lake at the bottom of the crater. If the sulphur is heaped up in a mound in the pot the flame is liable to go out.

Upon the principle of not putting all our eggs in one basket, it is best to have a number of pots when a large compartment is to be fumigated. A pot should contain not more than 30 pounds of sulphur, and the pots should be well distributed in various portions of the place to be disinfected.

Use 5 pounds per 1,000 cubic feet where a germicidal action is desired, and at least 2 pounds per 1,000 cubic feet for insecticidal purposes. For the destruction of bacteria an exposure of from 6 to 24 hours is necessary, while for the destruction of vermin from 2 to 12 hours is sufficient, depending upon the size and shape of the compartment to be treated.

Liquid Sulphur Dioxid.—Liquid sulphur dioxid, commonly known as sulphurous acid gas, while efficient, is about ten times as expensive as burning sulphur by the pot method. It has the advantage of liberating a large volume of the gas rapidly, thereby facilitating its dispersion. Further, the use of liquefied sulphur dioxid has the advantage of avoiding the danger of accidental fire.

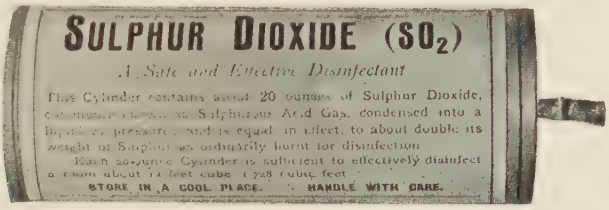


Fig. 156.—Liquefied Sulphur Dioxid in Tin Can.

One pound of the sulphur will produce about 2 pounds of sulphur dioxid: $S(32) + O_2(32) = SO_2(64)$. Therefore 2 pounds of the liquid sulphur dioxid is necessary to produce the same volume of sulphur dioxid as is generated from one pound of the burning sulphur.

The method of using the liquid sulphur dioxid is very simple. If the substance is bought in small tins it is only necessary to cut the lead pipes in the tops of the necessary number of cans and invert the latter in an ordinary washbowl or iron pot, when volatilization rapidly occurs. All the cans must be cut simultaneously and the operator must

act quickly and be prepared immediately to leave the room and shut the door. If the substance is contained in glass or metallic siphons the necessary amount of liquid sulphur dioxid can be projected from the outside through a pipe passed through the keyhole or other aperture. A suitable receptacle should be arranged on the inside to catch the drip and frozen mass which forms as a result of the expansion. In order to obtain the maximum disinfecting power with this method it is necessary to introduce moisture. This may be done by placing open pans of boiling water in the room, by injecting steam, or by a fine spray of water.

The Sulphur Furnace.—The sulphur may be burned in an apparatus of special construction known as a sulphur furnace, from which the resulting fumes are blown through a system of pipes into the room or hold of a vessel to be disinfected. Two forms of sulphur furnace are used: (1) the Kinyoun-Francis furnace, and (2) the Clayton furnace.

This method requires expensive and cumbersome machinery and has little to recommend it over the simpler pot method except that a large percentage of the gas may be blown into a given space. The pot method at best cannot produce an atmosphere containing more than 4 per cent. of sulphur dioxid, whereas it is theoretically possible to charge a confined space with a higher percentage of the gas by means of the furnace. In practice this is not possible without burning a great excess of sulphur and by expending a very long time, for the reason that the fumes first entering mix with the air and as the gas continues to flow into the space it displaces about an equal quantity of this mixture of sulphur dioxid and air, so that, as a matter of fact, in actual practice only about 2½ to 6 per cent. of the gas is usually obtained in the holds of vessels by the sulphur furnace.

It is advisable in using the sulphur furnace to arrange the pipe admitting the gas into the room as near the floor as possible. In disinfecting the holds of vessels the pipe is usually let down the hatchway until it is near the bilge. The heavy gas collects at the bottom and gradually ascends, displacing the air, so that it is important to allow an opening of some sort for the exit of the air near the top of the compartment that is being disinfected. This opening should not be closed until the gas escapes freely.

The *Kinyoun-Francis furnace* consists of an iron pan upon which the sulphur is burned. Under this pan is a firebox with ashpit and necessary drafts. The firebox is designed to hold a light fire of wood or shavings and is intended to heat the sulphur pan sufficiently to ignite the sulphur when thrown upon it at the beginning of the operation. This part of the apparatus is unnecessary, as the sulphur may be ignited more simply by means of some alcohol, turpentine, or kero-

sene on waste, or a few live coals. When once lighted there is no trouble in keeping the sulphur burning.

The air enters at A, Fig. 157, through a valve arranged to regulate the amount of flow. It then passes over the burning sulphur in the

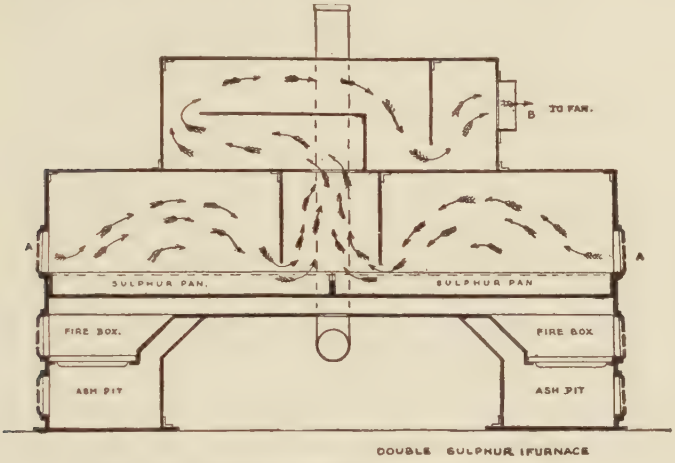


Fig. 157.—Section Through Sulphur Furnace.

direction shown by the course of the arrows to the fan. Fumes are compelled to take a devious course around the baffle plates and angle irons, as shown in the drawing, in order to insure complete combustion and to arrest sparks. From B the fumes are sucked to the fan, which is actuated by a steam engine or electric motor, and which forces the gas through the pipes to the place to be disinfected.

Running the fan at too high a speed may cause overheating of the pipes or the carrying over of sparks of burning sulphur. The proper amount of air should be carefully regulated so as to obtain complete combustion and the maximum amount of sulphur dioxid gas.

The pipe conducting the fumes from the sulphur furnace to the compartment to be disinfected gives a certain amount of trouble. It is apt to become clogged with sulphur which sublimes in the cooler parts. Ordinarily this pipe must be from 6 to 8 inches in diameter and may be made of smooth galvanized iron and the joints made tight with several layers of canvas saturated and coated with some fireproof paint. Rubber hose of this size is very expensive and soon vulcanizes.

No arrangement is made in this form of apparatus for adding water vapor to the sulphur fumes, which is necessary to obtain germicidal action. As a rule the holds of wooden vessels, in which sulphur fumigation is so much used, are usually so damp that the addition of more moisture is not necessary.

The *Clayton furnace* is a more compact apparatus than that just described. The sulphur dioxid is passed through a series of tubes sur-

rounded by water, an arrangement corresponding in all respects to the tubular condenser of a low-pressure steam engine. The Clayton furnace is furnished with a Root blower, and has the advantage that a comparatively large volume of sulphur dioxid may be pumped rapidly through pipes of small caliber without fear of overheating or fire. These furnaces are being installed upon ships for the purpose of fumigation at port and during the voyage for the destruction of rats, mice, and vermin. It is also an efficient fire extinguisher.

A portable sulphur furnace is a useful apparatus in municipal work, particularly in the fumigation of sewers, warehouses, stables, barns, and similar large, rough structures infested with vermin. This form of furnace was used with success in the fight against rats in the sewers of San Francisco in the anti-plague campaign.

Hydrocyanic Acid Gas.—Hydrocyanic acid gas is a very powerful insecticide, but a weak germicide. It appears to be effective against organisms no hardier than those of diphtheria and typhoid. On account of its extremely poisonous nature it has a very limited place in practical public health work for the destruction of bacteria. Hydrocyanic acid gas is useful in the treatment of stables, granaries, outhouses, compartments of ships, sleeping-cars, day coaches, and similar isolated or uninhabited places for the destruction of vermin. For its use as an insecticide see page 194.

Chlorin.—Chlorin is a germicide of considerable but uncertain power. It has little practical usefulness owing to its poisonous and destructive action. Both in its free state and its watery solution it has active deodorizing properties. Moisture is necessary for the disinfecting action of chlorin gas. At best chlorin, like all gases, is but a surface disinfectant.

Chlorin is an extremely irritating gas, and great care must be observed in its employment, for the inhalation of very weak proportions of the gas produces serious irritation, resulting in spasm of the larynx, bronchitis, and even in death. Chlorin is heavier than air (sp. gr. 2.47) and tends to fall. Therefore the vessel generating the gas should be placed in an elevated position in order to obtain anything like effective diffusion. Carpets, curtains, and fabrics generally are injured by its action, and the element is a very active bleaching agent.

The germicidal action of chlorin depends upon its great affinity for hydrogen. So strong is this affinity that it combines with the hydrogen of water in the presence of light, liberating the oxygen in its nascent state, thereby enabling the oxygen to exert its power upon organic matter. The value of chlorin as a deodorant depends upon its power of decomposing the offensive gases of decomposition such as sulphuretted hydrogen and volatile ammoniacal compounds.

The most convenient method of generating chlorin gas is by decom-

posing 1½ pounds of chlorid of lime with 6 ounces of strong sulphuric acid. This produces sufficient gas for the disinfection of 1,000 cubic feet of air space, or the gas may be generated from:

Common salt	8	ounces
Manganese dioxid	2	"
Sulphuric acid	2	"
Water	2	"

The following reaction takes place:

$$2NaCl+MnO_2+2H_2SO_4=Na_2SO_4+MnSO_4+2H_2O+Cl_2$$

Mix the water and the acid together and then pour the mixture over the salt and manganese dioxid in a glazed earthenware basin. The basin should rest on sand or in water.

Fisher and Proskauer have shown that in ordinary dry air 5.38 parts of free chlorin per 1,000 cubic feet of air space appear to be necessary to kill microorganisms. If the air is moist only 0.3 per cent. by volume in each 1,000 cubic feet of air is sufficient, disinfection being completed in 5 to 8 hours.

Free chlorin is much less useful than sulphur dioxid, since it is more difficult to control, more dangerous to manipulate, and more destructive in its effects.

Oxygen.—The disinfecting power of oxygen depends largely upon the physical state in which it exists. For instance, oxygen in the air has comparatively feeble germicidal properties when compared to nascent oxygen or ozone. The germicidal action of oxygen depends upon its very active property of combining chemically with the albuminous matter of the cell protoplasm. The oxidizing properties of this element partly explain the purifying action of fresh air. While most bacteria require the free oxygen of the air for their growth and multiplication, there is a large class of organisms (the anaerobes) to which the oxygen of the air acts like a poison or strong antiseptic.

Ozone.—Ozone is an allotropic form of oxygen containing three atoms of that element instead of two. In sufficient concentration it is a powerful germicide and has lately been found of practical use in the sterilization of water on a large scale for the use of cities and towns. It has also been used for the sterilization of bandages and other objects. There is not sufficient ozone in the air normally to exert any appreciable oxidizing or disinfecting properties. It requires at least 13 parts per million in the atmosphere to exert a definite effect upon bacteria; even then the action is not penetrating. Such quantities are harmful to man. (See page 585.)

LIQUID DISINFECTANTS

These consist of substances either in solution or suspension. An enormous number of such disinfectants have been exploited, but to be of practical value they must not only be strongly germicidal, but must also meet the many exacting requirements of general practice. Such substances are few in number.

Almost any chemical substance under one condition or another has the power to retard the development or destroy the activity of microbial life. We need only mention the well-known power of common salt or of sugar, both of which in sufficient concentration prevent the processes of fermentation and putrefaction. In weaker dilutions these same substances, on the contrary, favor growth of almost all the known bacteria.

The undeserved reputation of many chemical substances depends more upon their vile odor or judicious advertising than upon actual efficiency. Only those substances that have proven their worth by scientific tests and shown themselves to be trustworthy in actual practice will be discussed.

There is a complete analogy existing between a chemical reaction and disinfection, one reagent being represented by the disinfectant and the other by the protoplasm of the bacterium. Chick states that the velocity of disinfection increases with the rise in temperature in a manner similar to that of a chemical reaction. In fact, the temperature so greatly influences the disinfecting power of liquids that it is strongly recommended always to use warm solutions in actual practice. Even slight changes of temperature may make a great difference. Feeble antiseptic solutions become strong germicides when warmed. A good instance of the effect of temperature is given by Heiden,[1] who found that anthrax spores which survived the effects of a 5 per cent. carbolic solution for 36 days at room temperature were destroyed in half an hour in the same solution at 55° C. At 75° C. it took only 3 minutes to kill them. A 3 per cent. carbolic acid solution killed the same spores at this temperature in 15 minutes and a 1 per cent. solution in from 2 to 2½ hours.

It is not enough in applying any agent whose best working strength is known to use a small volume of the solution of that particular strength. There must be a sufficient amount of the substance used so that it shall be present throughout the whole mass in the proportion required. Thus an agent that is effective in a 2 per cent. solution cannot be used in that strength to disinfect an equal volume of an infected liquid, since the mixture would then contain but 1 per cent.

[1] *Centralbl. f. Bakt.*, Bd. 9, p. 221, 1891. *Archiv f. Hgy.*, Bd. 15, 1892.

Time is an essential factor too frequently disregarded in disinfecting with liquids in suspension or solution. Very few chemical disinfectants act instantly, even in strong solutions and under favorable conditions. The microorganisms are so often in clusters or are surrounded by mucoid films or imbedded in organic matter that no inconsiderable time is required for the disinfecting solution to penetrate to the germ. If the microbes are dry it takes a certain time to wet them before the chemical can act. This and other factors must be added to the time actually necessary for the substance to destroy the life of the germ after it comes in direct contact with it.

The medium in which the germs exist also makes a great difference so far as the power of liquid disinfectants is concerned. Behring found, for example, that anthrax bacilli suspended in water are killed in a few minutes with bichlorid of mercury solution of the strength of 1 to 500,000. In bouillon it requires a strength of 1 to 40,000, while in blood serum, if the disinfection is to be accomplished in a few minutes, a strength of 1 to 2,000 is not always sufficient. Therefore in the presence of organic matter or filth stronger solutions and longer exposures are required.

As a rule an emulsion has greater germicidal power than a solution. Thus soapy and resinous emulsions of the phenols may accentuate the germicidal power of these substances. Chick and Martin[1] have observed that the particles of an emulsion or soapy preparation of the coal-tar acids exhibit active Brownian motion. The bacteria are considerably larger than the mean diameter of the emulsified particles. The bacteria may plainly be seen to be bombarded by these particles. In this way the bacteria are frequently brought into intimate contact with the undiluted particles of pure coal-tar acids. The maximum effect may therefore be obtained and the death of the bacteria is inevitable. Such a concentration is evidently impossible with substances in solution. The coal-tar acids in suspension act upon the bacteria through physicochemical absorption, and not through chemical combination. The bacteria rapidly become surrounded by the disinfectant in a much greater concentration than actually exists within the liquid. Other particulate matters present have the power of absorption, and their presence therefore interferes with the germicidal value of substances in emulsion. Thus the value of phenol is barely impaired by the presence of organic matter in solution, while emulsified disinfectants are reduced to one-third or one-half their original value.

Germicidal substances in emulsion have less power of penetration than substances in solution. The emulsified substances are deposited upon and adhere to the surface of the mass. This may readily be seen by adding one of the coal-tar emulsions to a fecal mass, in which case

[1] *Jour. of Hyg.*, Vol. VIII, No. 5, 1908.

a visible layer of the coal-tar creosotes may be seen to collect upon the surface. These facts emphasize the great importance of breaking up all masses requiring disinfection.

Chemical substances act in a great variety of ways to bring about the destruction of bacteria. Just how the microbes are poisoned is, in many instances, an unsolved problem in toxicology. In particular cases there appears to be a chemical union between the disinfectant and the protein of the bacteria, as appears to be the case with corrosive sublimate or formaldehyd. In some instances the mycoprotein of the cell is coagulated, as in the case of carbolic acid and homologous substances. It has been shown that the higher the grade of dissociation the greater is the disinfecting power of the solution. Thus in the case of the soluble metallic salts, and especially mercury, it depends upon whether in the electrolytic dissociation the metal exists as an independent ion or whether it exists as a complex ion. In the first case the solution has strong germicidal properties; in the second these properties are much weaker. In other liquids, as, for example, alcohol, ether, etc., the metallic salts have very slight dissociation which, according to Krönig and Paul, explains the weaker disinfecting power of these solutions. The disinfecting power of metallic salts depends, furthermore, not only upon the influence of the metal ion, but also upon the other ions and upon the unassociated parts of the metallic salts.

The reaction of the solution and of the medium to be disinfected varies with the substance employed. Thus lime is an alkali, and if used to disinfect an acid substance enough must first be added to neutralize the medium and then an additional amount of lime must be added necessary to accomplish the disinfection. In the same way, if mercuric chlorid is added to solutions containing sulphids, caustic alkalies, or certain metallic salts, sufficient must be added in order to first precipitate these substances and then enough more added to exert its disinfecting action. Likewise, the greater the number of germs to be destroyed the greater the amount of the disinfectant required to accomplish the purpose.

The choice of the chemical to be used depends somewhat upon the nature of the substance to be disinfected as well as upon the resistance of the virus. For example, bichlorid of mercury is inapplicable to the disinfection of albuminous matter. Certain chemicals have a selective action and appear to be specific poisons for some microorganisms. Taken altogether, therefore, the choice of the chemical, its strength, and time of application, the temperature of the liquid, and its method of employment are all problems which must be solved for each particular case.

Methods of Using Chemical Solutions.—There are various ways of applying chemical solutions for disinfecting purposes. No method is

trustworthy that does not thoroughly wet the object with the solution, so that there may be direct contact between the substance in solution and the contagious principle against which the process is directed.

As a rule this may best be accomplished by immersing the infected object in the solution. When this is not practicable the solution must be applied to the object. A favorite way of applying disinfecting solutions to surfaces, such as walls, ceilings, the holds of ships, and other rough structures, is by means of a hose. The pressure is supplied either by elevating the tank containing the solution or by means of a pressure pump. As bichlorid of mercury is practically the only disinfectant used in this way, the pump should be made of iron and have no copper, brass, or steel parts exposed to the corroding action of the bichlorid of mercury.

In applying the disinfecting solution to the surfaces of a room or the hold of a ship the operator should begin at one corner of the ceiling, wetting that first, and then go over every portion of the walls systematically, from above downward. The floor comes last.

Solutions thus applied remain but a short time in contact with the surfaces to be disinfected. It is therefore an advantage to have the solution hot and strong and to have sufficient pressure, in order to obtain the mechanical cleansing effect produced by a vigorous stream.

Another method of applying disinfecting solutions to surfaces is by means of mops, brooms, and the like.

The pulverizer is very popular in France for the disinfection of walls and other surfaces with solutions of bichlorid of mercury. The apparatus for this purpose consists of a metal cylinder fitted with a simple force pump which compresses the air in the reservoir. The solution does not come in contact with the pump. The current of air driven through one tube sucks the solution through the other and sprays it from the nozzle in a nebulous cloud, similar in principle to the well-known hand atomizers. It is easy to demonstrate, by using a colored solution upon a white wall or sheet, that a liquid sprayed in this way does not wet the entire surface. The method is therefore an unscientific and unreliable one when used with a non-volatile chemical.

Bichlorid of Mercury.—$HgCl_2$, bichlorid of mercury or mercuric chlorid, commonly called corrosive sublimate, is one of our most valuable and potent germicides. It destroys all forms of microbial life in relatively weak solutions. It kills both germs and their spores. It is not a deodorant.

The disadvantages of bichlorid of mercury are that it corrodes metals, forms insoluble and inert compounds with albuminous matter, and is very poisonous. These disadvantages place distinct limitations upon its use.

Mercuric chlorid ($HgCl_2$) is a white, crystalline substance of heavy specific gravity (5.43). It volatilizes somewhat more readily than mercurous chlorid (calomel), even at room temperature. On account of this property caution should be observed to remove bichlorid solutions from living-rooms, some instances of poisoning having been traced to this neglect. It is therefore well to follow bichlorid with clear water and a cleansing is always in order.

Bichlorid of mercury will dissolve in 16 parts of cold water and 3 parts of boiling water. As it is not readily soluble in water, it is convenient to keep a saturated alcoholic solution on hand and use this to make the watery solution. A 25 per cent. solution may readily be made in alcohol, and by the addition of hydrochloric acid or ammonium chlorid this solution keeps well without precipitation. As this method would be rather expensive for making up the large quantities required in flushing the holds of ships or other extensive surfaces a little device pointed out by Geddings will be found serviceable. This consists in weighing out the correct quantity of the bichlorid, which is placed in a canvas bag, and this is hung from the faucet so that the water will run through it into the tank or receptacle holding the solution.

The solution of bichlorid of mercury is facilitated by the presence of hydrochloric acid or a chlorid such as ammonium chlorid or common salt. Twice the weight of these substances should be added to the quantity of bichlorid used. If the solution is to be pumped or otherwise come in contact with metals it is better to use the salt than the acid, because the acid solution of bichlorid is very destructive to the metal parts of the pump and to the couplings and nozzle of the hose, particularly if this is made of copper or brass. Sea-water contains about 4 per cent. of salt, and is well suited for making bichlorid solutions. It is extensively used at seaport quarantine stations for this purpose.

Laplace first pointed out that the addition of a small amount of an acid to the solution of bichlorid of mercury greatly increases its efficiency, and by lessening the formation of insoluble albuminates also increases its power of penetration. This was later denied by Krönig and Paul, who assert that the addition of sodium chlorid to a watery solution of bichlorid diminishes its power. They found that potassium chlorid or hydrochloric acid has the same effect.

The germicidal action of bichlorid solution seems to depend upon the reaction which takes place between the salt of mercury and the mycoprotein of the germ. Geppert has shown that in the reaction which takes place between the bichlorid of mercury and the spores of anthrax the vitality of the latter may seem to be lost, but that the bichlorid may be precipitated from its combination by the action of ammonium sulphid, which restores the viability of the spore.

Bichlorid of mercury is decomposed by lead, tin, copper, and other metals, and therefore should not be made or kept in metal receptacles. Lead pipes are rendered brittle and worthless. Care must therefore be exercised in using this solution about water-closets and house plumbing.

Corrosive sublimate is precipitated in alkaline fluids containing albuminous substances. The precipitate consists of insoluble and inert compounds; therefore corrosive sublimate should not be used for the disinfection of media containing much organic matter, particularly when the reaction is alkaline. It is totally inapplicable to the disinfection of sputum and feces, for it forms a coagulum which prevents the further penetration of the bichlorid. It also unites chemically with sulphids and the caustic alkalies, so that it should not be employed as a disinfectant when these substances are present in any considerable amount.

To diminish the danger from accidents in households and hospitals bichlorid solutions should be colored with permanganate of potash or indigo or one of the anilin dyes.

Bichlorid of mercury is usually used in the proportion of 1 to 500 or 1 to 1,000. A solution of 1 to 1,000 is ample for the destruction of all the non-spore-bearing bacteria, provided the exposure is continued not less than half an hour. Many bacterial cells are killed almost at once when brought into direct contact with a solution of this strength, and the great majority perish within 15 minutes. The extra time allows for penetration and provides a factor of safety. Warm solutions are much more potent than cold. For spores a solution of 1 to 500 is necessary and an exposure of not less than one hour.

For practical work the solution may be made as follows:

Corrosive sublimate	1 dram	1 gram
Water	1 gallon	1 liter

Mix and dissolve.

This is approximately a 1 to 1,000 solution. One ounce of this solution contains very nearly half a grain of corrosive sublimate.

Carbolic Acid.—Carbolic acid is a very useful disinfecting substance with a wide range of application. It should not be depended upon to kill spores. As it does not coagulate albuminous matter as actively as corrosive sublimate it may be used for the disinfection of soiled clothing and bedding, as well as for excreta and sputum.

Carbolic acid is a popular term for an ill-defined mixture consisting largely of phenol and phenolic bodies. Phenol has the chemical structure of an alcohol; it is represented by the formula: $C_6H_6O = C_6H_5OH$. It is produced in the dry distillation of coal, and is the chief

constituent of the acid portion of coal-tar oil. Pure phenol crystallizes in long, colorless needles. Commercial phenol forms a crystalline mass, which is apt to turn reddish in time and in contact with moist air deliquesces to a brownish liquid. Carbolic acid has a penetrating odor and a strong burning taste and is a corrosive poison.

The carbolic acid of commerce contains cresols and higher homologs, some of which have a higher germicidal value than pure phenol itself. The commercial product also contains tar oils which are totally lacking in bactericidal properties. It should be remembered that the crude carbolic acid has a higher germicidal potency than the pure phenol.

Carbolic acid dissolves in water with some difficulty and should therefore be thoroughly mixed. At ordinary temperatures phenol is soluble in about 15 parts of cold water; that is, a saturated solution contains between 6 and 7 per cent. Carbolic acid or phenol is commonly used in solutions of 2.5 to 5 per cent., which are entirely trustworthy for destruction of all infectious processes due to non-spore-bearing organisms. Warm solutions are much more potent than cold.

Carbolic acid when dissolved in alcohol or ether loses in germicidal value; the addition of 0.5 per cent. of hydrochloric acid aids its activity.

McClintock and Ferry[1] have shown that the large majority of the coal-tar disinfectants (carbolic acid, cresols, and the like) do not destroy the virulence of vaccine virus in one-half per cent. solutions at five hours' exposure, while with this strength and length of time these disinfectants would destroy practically all non-spore-bearing bacteria. The inference, therefore, is allowable that this class of disinfectants is not safe to use for such diseases as smallpox and the presumably protozoal diseases such as syphilis, measles, scarlet fever, etc.

The fact that carbolic acid and phenol do not actively coagulate albuminous matter renders them suitable to the disinfection of excreta and organic matters generally. They are not destructive to fabrics, colors, metals, or wood in the strengths used, and therefore may be employed for the disinfection of a great variety of objects. Crude carbolic acid, although it has a stronger germicidal power than pure phenol, has the disadvantage of having a more pungent and penetrating odor and leaves a deposit of coal-tar oils and other impurities.

There has been much disparagement of carbolic acid because laboratory tests have clearly demonstrated that it cannot be depended upon to kill spores. This limits but does not destroy its usefulness, especially as the great majority of the epidemic diseases of man are due to non-

[1] McClintock, Chas. T., and Ferry, N. S.: "The Resistance of Smallpox Vaccine to the Coal Tar Disinfectants," *Jour. of the Am. Pub. Health Assn.*, Vol. I, No. 6, June, 1911, pp. 418-420.

spore-bearing bacteria. The time of exposure to a 3 or 5 per cent. solution should be not less than half an hour. Fabrics are usually immersed for one hour.

The Cresols.—By far the majority of the disinfectants sold to the public are mixtures of varying quantities of phenolic bodies with inert tar oils and an emulsifying agent such as soap or tar, and sometimes resin, gelatin, or dextrin. These substances all possess a smell distinctive of carbolic acid and are effective germicides. The cresols, $C_6H_4(CH_3)OH$, have the advantage over carbolic acid or pure phenol in that they readily form beautiful emulsions, have a higher germicidal value, and are less poisonous. It has already been pointed out that substances in emulsion are more potent germicides than solutions.

Cresol.—Cresol is prepared from coal-tar by collecting the distillates coming over between 140° C. and 220° C., and then purifying these distillates by treatment with solution of sodium hydroxid and hydrochloric acid. Cresol is a mixture of the three isomeric cresols obtained from coal-tar and freed from phenol, hydrocarbons, and water. It is also known as cresylic acid and trikresol.

Creosote.—Creosote is a highly refractile liquid obtained from the destructive distillation of wood or coke. Wood-tar creosote for medicinal use is obtained from beachwood; it is a complex mixture of phenoloid bodies, the proportions of which differ according to the modes of distillation and purification. It contains phenols, cresols, and higher homologs. Coal-tar creosote, sometimes called creosote oil, contains that portion of the distillate from coal-tar intermediate between crude naphtha on the one hand and pitch on the other. Coal-tar creosote contains phenols, cresols, and higher phenoloid bodies, also naphthalene and other solid hydrocarbons, as well as pyridin and other bodies of basic character. Creosotes vary in composition, and owe their germicidal properties to the phenol and cresols which they contain. They are seldom used as such, but form bases of many commercial disinfectants after purification or the addition of alkalies or soaps. It is the creosote from coal-tar, and not wood-tar, that is used as a germicide in public health work.

Trikresol.—Trikresol consists of a mixture of ortho, meta, and paracresols. Matacresol is a liquid; the other two are solid crystalline bodies having a low melting point. These cresols are some of the impurities found in commercial carbolic acid. The cresol group forms the next higher homolog to phenol, one atom of hydrogen being replaced in the latter by the methyl radical, CH_3. The cresols are very insoluble in water. Their solution may be facilitated by the use of alcohol or glycerin. Trikresol is a clear or pink-colored syrupy liquid. It is soluble to the extent of about 2½ per cent. in water. It is somewhat less

poisonous than carbolic acid; its uses are the same. It is an effective germicide in a 1 per cent. solution.

Liquor Cresolis Compositus.—Liquor cresolis compositus of the U. S. Pharmacopœia consists of cresol, 500 gm.; linseed oil, 350 gm.; potassium hydroxid, 80 gm.; and water sufficient to make 1,000 gm. This officinal mixture makes a clear solution in water. The solution is intended as a substitute for the many commercial preparations of cresol on the market. It has practically the same uses as the trikresol of commerce.

Creolin.—Creolin is an emulsion of cresols and certain other products contained in crude carbolic acid with rosin soap. Creolin forms a milky emulsion when mixed with water. It is sometimes called cresolin or sanatol. At least two sets of preparations are on the market: one of German, the other of English origin. It is used in 1 or 2 per cent. solution.

Lysol.—Lysol is a brown, oily-looking, clear liquid with a creosote-like odor. It is similar to creolin except that it has more of the cresols and less of the other products. It is made by dissolving the fraction of tar oil which boils between 190° and 200° C. in fat and subsequently saponifying by the addition of alkali in the presence of alcohol. It contains 50 per cent. of cresols, is miscible in water, forming a clear, saponaceous, frothy liquid. It is more powerful as a germicide than phenol, and is usually used in 1 per cent. solution.

Solveol and Solutol.—Solveol is a solution of sodium cresolate in excess of cresol. Solutol is a solution of cresol in excess of sodium cresolate.

There are a vast number of other commercial disinfectants of similar nature consisting of coal-tar creosotes in combination with alkalies, soaps, resins, etc., such as chloronaphtholeum, sulphonaphthol, bacillol, paracresol, and other trade names.

Formalin.—Formaldehyd in solution is known as formalin. This is a very valuable disinfectant with a wide range of usefulness in general practice. It is superior to bichlorid of mercury for many purposes, especially as its action is not retarded by the presence of albuminous matter. Formalin is not injurious to most articles, and it is not very poisonous. It is a true deodorant. The more I work with formaldehyd, both in solution and as a gas, the more am I impressed with its trustworthiness.

Formalin consists of a 40 per cent. solution of the gas formaldehyd (HCHO) dissolved in water. The liquid is a clear solution, giving off an appreciable odor of the gas. It is exceedingly irritating, but not especially toxic. Formalin solutions are rather unstable. There is a constant loss by evaporation if the liquid is not kept well corked, and in cold weather the formaldehyd polymerizes and precipitates in one of

its polymeric forms—trioxymethylene. For the description and discussion of formaldehyd see page 993.

Hot formalin attacks iron and steel, and therefore is not suitable. It does not attack copper, brass, nickel, zinc, and other metal substances. It causes no diminution in strength of textile fabrics and has no bleaching or other deleterious effects upon colors. Formalin renders leather, furs, and skins brittle as a result of the union that takes place between the formaldehyd and the organic matter of these articles, and they should therefore be disinfected by another process.

A 10 per cent. solution of formalin in water is about the equivalent of a 1 to 500 solution of bichlorid of mercury, or superior to a 5 per cent. solution of carbolic acid. It must be borne in mind that in speaking of a solution of formalin a solution is meant of the liquid containing 40 per cent. formaldehyd; that is, a 1 per cent. solution of formalin would contain that liquid in proportion to 1 to 100, but would contain the substance formaldehyd in the proportion of 1 to 250.

Fecal masses are deodorized almost instantly by a small quantity of formalin, and are disinfected in a short time when mixed with an equal volume of a 10 per cent. solution. It is advisable to continue the contact one hour to insure complete action.

There is some discrepancy as to the percentage of formalin solution necessary to accomplish trustworthy disinfection in general practice. Taking into account the deterioration of the solution with age and allowing an excess as an element of safety, it is recommended that at least a 5 per cent. solution be used, but in the presence of organic matter 10 per cent. is recommended. It may be used to disinfect urine, excreta, sputum, and other similar substances.

Potassium Permanganate.—Potassium permanganate is a germicide of undoubted value, but of very limited application in general practice on account of the readiness with which it is reduced and rendered inert by organic matter. Despite its limitations it ranks high on the list of germicides for certain definite purposes, more particularly in surgical practice. It has been much used in India and other places for the purification of water.

Potassium permanganate ($K_2Mn_2O_8$) is a dark purple, crystalline substance with a sweet, astringent taste. A few crystals impart to a large quantity of water a rich purple tint which is destroyed by organic matter and deoxidizing agents. It is soluble in 16 parts of cold and 2 parts of boiling water. The stain produced by potassium permanganate may be removed by a solution of oxalic acid, muriatic acid, or simple lemon juice.

Potassium permanganate readily gives up its available oxygen, and it is the free nascent oxygen that is the true disinfecting agent. Sternberg found a solution of 1 to 833 sufficient to kill pus cocci in two hours.

Koch found that a 5 per cent. solution killed spores in one day. Loeffler found that the bacillus of glanders is destroyed in two minutes by a 1 per cent. solution.

Water containing organic matter may be purified to a certain extent and rendered palatable by adding, drop by drop, a solution of permanganate until the pink color of the water ceases to be destroyed after the lapse of 24 hours. The clear liquid may then be decanted and used. Permanganate used in this way does not reach sufficient concentration to be a trustworthy germicide.

Lime.—Lime in certain of its variable chemical combinations is the best and cheapest disinfecting substance we have. It is usually used either as lime or chlorinated lime.

Lime, or quicklime, is a very caustic substance used for the destruction of organic matter as well as germ life. On account of its efficiency and cheapness it is a valuable addition to the list of practical disinfectants. Lime or calcium oxid (CaO) is one of the alkaline earths. It is not so caustic as the alkalies, having less affinity for water. It is obtained by calcining native calcium carbonate ($CaCO_3$), such as chalk, limestone, or marble, by which the carbon dioxid is driven off and the calcium oxid remains behind. Lime as such requires the addition of water for germicidal purposes.

Slaked Lime.—Slaked lime or calcium hydroxid, $Ca(OH)_2$, is prepared by adding one pint of water to two pounds of lime. The lime absorbs about half its weight of water. The mass becomes heated and the air escapes from the pores of the lime with a hissing noise. The result is calcium hydroxid or slaked lime. Upon exposure to the air the slaked lime will absorb still more water and also carbon dioxid, converting it into calcium carbonate, which is inert so far as its disinfecting power is concerned. Freshly slaked lime should therefore always be used.

Whitewash is slaked lime mixed with water. It is commonly used for the disinfection, sweetening, and brightening of the walls of cellars, rooms, barracks, barns, stables, poultry-houses, and out-buildings generally. Whitewash is a very satisfactory method of destroying spore-free bacteria that may have lodged upon such surfaces. A mordant such as glue is usually added to whitewash to make it adhere.

Milk of lime is slaked lime mixed with about four times its volume of water to the consistency of a thick cream. It is useful for the disinfection of excreta and privy vaults. Air-slaked lime containing the inert carbonate must not be used in the preparation of whitewash or milk of lime, freshly slaked lime containing calcium hydroxid being necessary to accomplish disinfection. Calcium hydrate is mostly insoluble and settles to the bottom; the milk of lime must therefore be agitated to restore its homogeneous character before it is used. Milk

of lime is most powerful when freshly prepared. It soon changes to the inert carbonate, and therefore should not be used if more than a few days old unless carefully protected from contact with the air.

Almost all laboratory experiments, while differing somewhat in certain unimportant particulars, confirm the conclusions of the early investigators as to the great practical value of lime as a germicide. A 1 per cent. watery solution of the hydroxid kills non-spore-bearing bacteria within a few hours. A 3 per cent. solution kills typhoid bacilli in one hour. A 20 per cent. solution added to equal parts of feces or other filth and mixed with them will completely sterilize them within one hour.

Lime is particularly valuable in the disinfection of excreta. The lime in one form or another must be well incorporated with the mass and enough must always be added in order to make the reaction of the mixture distinctly alkaline. Sternberg recommends that freshly prepared milk of lime should contain about one part by weight of hydrate of lime to eight parts of water. This should be used freshly prepared and added in quantity equal in amount to the material to be disinfected. The mixture should be allowed to stand at least two hours before final disposal. Fortunately, this valuable disinfecting agent is very cheap, so that it can be used with a liberal hand in excess of the amount which scientific tests find necessary.

Lime has been used in very early times in connection with the disposal of the dead. The method is an admirable one for the burial and disinfection of bodies dead from a communicable disease. The body should be placed in a tight coffin with twice its weight of fresh, unslaked lime, without the addition of water or moisture in any form.

Chlorinated Lime ("Chlorid of Lime").—Chlorinated lime was used as a disinfectant and deodorant long before bacteriology was a science. The early work of Sternberg demonstrated that the confidence placed in this substance from an empiric standpoint is justified by scientific tests. Chlorinated lime under certain circumstances, in fact, is one of the most powerful germicides we possess, and has been used particularly for the disinfection of sewage and water.

Chlorinated lime, popularly miscalled chlorid of lime, is a soft, white, friable substance, and is known also as bleaching powder. It has a peculiar chemical composition and is somewhat unstable. It is made by passing chlorin gas through lime. Owing to its affinity for moisture, which it slowly absorbs from the air, it soon becomes pasty and loses some of its chlorin; the hypochlorites are reduced to chlorids, which are inert as germicides. Freshly prepared chlorinated lime should have a very slight odor of free chlorin. A strong odor of this gas indicates that deterioration of the substance is taking place. It

should therefore only be used when freshly prepared and when kept in air-tight receptacles.

Chlorinated lime is made by passing nascent chlorin gas over very slightly moist calcium hydroxid. Concerning its exact chemical composition there is some disagreement. It is represented by the formula $CaOCl_2$ or ClCaOCl or Ca(ClO)Cl. According to the U. S. Pharmacopœia it should contain not less than 35 per cent. of available chlorin. The British standard is 33 per cent. and the German 25 per cent. Chlorinated soda has almost the same germicidal value as chlorinated lime. Chlorinated soda is sold only in solution, and is prepared by mixing a solution of chlorinated lime and sodium carbonate.

Chlorinated lime is only partially soluble in water or in alcohol. A solution in water of 0.5 to 1 per cent. will kill most bacteria in one to five minutes. A 5 per cent. solution usually destroys spores within an hour.

While the solution of chlorinated lime has an indefinite composition it is generally admitted to contain calcium hypochlorite ($CaClCaClO_2$), which is its active disinfecting principle. It also contains calcium chlorid ($CaCl_2$), which has a great affinity for water, and calcium hydrate ($Ca(OH)_2$), which is largely insoluble. The calcium hypochlorite, upon which the efficiency of the solution largely depends, is readily broken up, even by the carbon dioxid found in the air and water, into hyperchlorous acid, and this acid is so unstable that even in the presence of light it is decomposed into hydrochloric acid and free chlorin, both of which are active germicides. When bleaching powder is added to water it is the nascent oxygen, and not the chlorin, that is the disinfecting agent. (See page 797.) The solution is highly alkaline and has distinct bleaching powers. Its action as a deodorant depends not only upon its destructive influence upon organic matter and its germicidal properties, but also upon its great affinity for water, thus acting as a disinfectant. It also has the power of combining with hydrogen sulphid and the volatile ammoniacal compounds of decomposition and decay.

Chlorinated lime not only bleaches but is destructive to fabrics. If the solution is employed for the disinfection of body linen and washable clothing these articles must, after a not too long immersion, be thoroughly washed in plenty of fresh water.

It should be remembered that the hypochlorites are decomposed and practically rendered inert by organic matter. They should therefore be used largely in excess. Thus a preparation containing 10 per cent. of available chlorin has the high carbolic coefficient of 21.0, but on mixing an equal amount of this preparation with urine and allowing the mixture to stand one hour the coefficient falls to 0.8 per cent.

(Klein).[1] Gruber points out that the efficiency of chlorinated lime, when used to disinfect cattle wagons, is greatly increased by first thoroughly washing away the organic matter.

Chlorinated lime may be used either as a dry powder or in solution. As a dry powder it is very generally used by strewing it into damp corners of cellars, privies, and similar places, where it acts as a deodorant and desiccant. The dry substance may also be used to disinfect excreta. For this purpose enough of the chlorinated lime must be added and well incorporated with the mass to make a 4 or 5 per cent. solution.

In the U. S. Army a 4 per cent. strength of chlorinated lime in solution is officially prescribed for use in the disinfection of the excreta of the sick, it being specifically stated that the chlorinated lime so used shall be of good quality and not have undergone decomposition. A solution known as the "American standard," containing 6 ounces of the powder to the gallon, is largely used for the disinfection of discharges and for the scrubbing of floors and other surfaces. In recent years chlorinated lime or chlorinated soda has come into special prominence on account of its use for the disinfection of drinking water. A surprisingly minute amount will disinfect a large volume of water. The amount required depends upon the quantity of organic matter contained in the water. A reasonably clean water may be rendered practically sterile by the addition of 0.1 of a part of chlorinated lime (estimated as available chlorin) to 1,000,000 parts of water. For waters containing organic matter as much as 1 to 5 parts per 1,000,000 may be required. (See page 797.)

A convenient method for using chlorinated lime to disinfect drinking water is to add 1 gram of chlorinated lime containing approximately 30 per cent. of available chlorin to 1 liter of water. This should be mixed thoroughly and enough of the mixture added to the water in question to make one part of chlorinated lime to 200,000 parts of water. This should be allowed to stand at least 20 minutes after having been thoroughly shaken, and the water may then be regarded as safe, so far as typhoid, cholera, and similar infections are concerned.

Chlorinated lime may also be used to advantage to disinfect the bath water in cases of typhoid fever, dysentery, cholera, or other communicable diseases. It may also be used for the disinfection of springs, wells, cisterns, tanks, etc.

The Hypochlorites.—Labarraque's Solution.—Labarraque's solution is an aqueous solution of several chlorin compounds, chiefly sodium hypochlorite (NaClO) and sodium chlorid (NaCl), and should contain at least 2.6 per cent. by weight of available chlorin as deter-

[1] *Public Health*, Oct., 1906. Confirmed by Rideal, Sommerville, Moore, and others.

mined by titration with thiosulphate. The solution is clear and colorless when pure. If prepared with an excess of chlorin it is yellowish in color. It has a feeble odor of chlorin and bleaches indigo, litmus, and vegetable dyes. In practice this solution diluted with water 1 to 4 is mainly used for the disinfection of the person, but as it is more expensive and somewhat less efficient than chlorinated lime it has no advantages over that substance.

Antiformin.—Antiformin is the patented name of a disinfectant which was introduced in 1900 by Victor Tornell and Axel Sjoo of Stockholm as a cleansing material for fermenting vats in breweries, but it is only since the investigations of Uhlenhuth and Xylander[1] in 1908 that it has come into prominence in bacteriological and sanitary work.

Antiformin consists of equal parts of liquor sodæ chlorinatæ of the British Pharmacopœia and a 15 per cent. solution of caustic soda. The formula for the liquor sodæ chlorinatæ is as follows:

Sodium carbonate	600
Chlorinated lime	400
Distilled water	4,000

Dissolve the sodium carbonate in 1,000 c. c. of the distilled water; triturate thoroughly the chlorinated lime in the remainder of the water; filter; mix the two and filter again.

Antiformin has a strong germicidal action in weak solutions (2 to 5 per cent.), killing ordinary cocci and some bacilli rapidly, five minutes at most being sufficient. In this respect antiformin acts more rapidly and surely than either of its component parts used alone. It has, however, very slight action upon the tubercle bacillus, the smegma bacillus, and other organisms belonging to the acid-fast group.

Antiformin is an almost colorless liquid, with a strong odor of chlorin, and is strongly alkaline. It keeps fairly well without particular precautions being taken. It has deep powers of penetration, owing to its ability to dissolve and render homogeneous the various substances in which bacteria are often found, such as sputum, feces, pus, urinary sediment, and even small pieces of tissue.

The germicidal action of antiformin is doubtless due to the energetic oxidizing properties of the chlorinated lime. The fact that it does not kill the tubercle bacillus and other acid-fast organisms seems to be due to the biochemical nature of these bacilli. The fatty or waxy capsule which is present and which gives them their acid-fast property acts as an impervious coat, resisting the dissolving action of the antiformin, and so protects the protoplasm of the bacilli from its

[1] *B. klin. Wochenschr.*, LXV, No. 29, July 20, 1908.

germicidal action. The tubercle bacillus may be isolated in pure culture by exposing tuberculous sputum to a 20 per cent. solution of antiformin for 24 hours at room temperature or 4 to 6 hours at incubator temperature. The bacilli may then be thrown down by centrifugalization, washed free of alkali, and then planted upon solidified egg or other suitable culture medium, or injected into susceptible animals.

While antiformin is therefore a very active germicide for the ordinary bacteria it cannot be depended upon for the acid-fast group.[1]

Bromin and Iodin.—Bromin and iodin have about the same value as chlorin, both in their gaseous state and in solution. The tincture of iodin is now much used in surgery for the disinfection of the skin.

Ferrous Sulphate.—Ferrous sulphate has long been valued as a disinfectant on account of its power as a deodorant, and has been used extensively, being a comparatively cheap substance. Its germicidal power has been shown by laboratory tests to be rather feeble, so that it cannot be depended upon as a trustworthy disinfectant.

Ferrous sulphate ($FeSO_4$), commonly called green vitriol, iron vitriol, or copperas, consists of large bluish-green crystals which slowly effervesce and oxidize in the air. It is soluble in about twice its weight of cold water, forming a greenish solution. It is a much less powerful germicide than the sulphate of copper, and is limited in use to the destruction of odors, and even for this purpose is not always successful.

Sulphate of Copper.—Sulphate of copper ($CuSO_4$) is about half as strong as bichlorid of mercury. It has a peculiar selective action in that it has a remarkable affinity for many species of algæ which are killed in the proportion of 1 to 1,000,000. Algæ are the most common cause of unpleasant odors and tastes in drinking water, and sulphate of copper may therefore be used to check or destroy their growth. (See page 800.) In these great dilutions sulphate of copper will not kill the typhoid bacillus, so that it is not practical to use it as a disinfectant in water.

Chlorid of Zinc.—Chlorid of zinc (ZnCl) was at one time highly valued as a disinfectant, and is still extensively used despite the fact that it stands rather low in the list of germicidal agents. It has even weaker powers as a disinfectant than ferrous sulphate and cannot be recommended as trustworthy. It has some value as a deodorant.

ACIDS

Acids in sufficient concentration are very effective germicides. An amount of acid which equals 40 c. c. of normal hydrochloric acid per

[1] Paterson, R. C.: "A Report on the Use of 'Antiformin' for the Detection of Tubercle Bacilli in Sputum, etc.," *Jour. of Med. Research*, Vol. XXII, No. 2, April, 1910, p. 315.

liter is sufficient to prevent the growth of all varieties of bacteria and to kill many. The variety of acid makes little difference. The mineral acids are more corrosive and also more germicidal than the vegetable acids. A 1 to 500 solution of sulphuric acid kills typhoid bacilli within one hour. Hydrochloric acid is about one-third weaker, and acetic acid somewhat weaker still. Citric, tartaric, malic, formic, and salicylic acids are similar to acetic acid. Boric acid destroys the less resistant bacteria in 2 per cent. solution and inhibits the others. (Park.)

SOAPS

Ordinary soaps have but limited disinfecting power. According to Behring the germicidal power of soaps depends upon their alkalinity, but Serafini more correctly points out that the free alkali present, even in concentrated soap solutions, is so small in amount that it can exert no disinfecting action whatever, and that neither the alkali nor the fatty acid, nor the combination of the two is the effective agent.

Unfortunately, the disinfecting power of soap solutions is not marked enough to make them trustworthy disinfectants despite their great value as detergents. The common commercial soaps, especially the colored soaps, are frequently of very poor quality, containing rosin instead of fat, and are not to be depended upon. The soft soaps should also be avoided on account of the presence of all the impurities of the fat and alkali from which they are made. There are other conditions which render the use of soaps uncertain, the chief of which is the hardness of the water.

The action of soap solutions is much influenced by the temperature, which is easy to understand when we recall the powerful germicidal action of hot water alone. It has been shown that soap, even in strong solution and with prolonged exposure, cannot be trusted to destroy the infection of typhoid, cholera, or the micrococci of suppuration. Therefore soaps alone cannot be depended upon for the disinfection of objects and clothing, but in conjunction with certain compatible chemicals, and also with the mechanical cleansing which always accompanies their application, soaps have a wide and varied usefulness.

Soap solutions should always be made with soft water. The addition of one of the caustic alkalies, as lye, increases their germicidal and detergent value. The solution should be strong, containing not less than 10 per cent. of soap, and the water should be as hot as possible and applied with mops or brushes.

Medicated soaps are for the most part a snare and delusion so far as any increased germicidal action is concerned. In fact, the addition of carbolic acid, bichlorid of mercury, and other substances which have the property of combining chemically with the soap seems actually

to diminish the disinfecting value of that substance. As a rule a very small quantity of the disinfecting substance is added to the soap, and when we call to mind what an exceedingly small quantity of soap is generally used for the ordinary washing of the skin and the further dilution of this small amount by the water used it is easy to understand that medicated soaps as ordinarily applied cannot have an energetic disinfecting action.

An exception seems to be the soap devised by McClintock, in which a mercury salt exists unchanged and active. He found that double iodid of mercury answers this purpose in the proportion of 0.05 to 2 per cent. A solution containing 1 per cent. of the soap was found by him to be fatal to pus cocci, cholera, diphtheria, and typhoid bacilli in one minute. This soap does not attack nickel, silver, aluminium, steel instruments, or lead pipes, and does not coagulate albumin.

The following is a table of comparative antiseptic values taken from Park:

TABLE OF ANTISEPTIC VALUES

Alum	1 : 222	Mercuric chlorid	1 : 14,300
Aluminium acetate	1 : 6,000	Mercuric iodid	1 : 40,000
Ammonium chlorid	1 : 9	Potassium bromid	1 : 10
Boric acid	1 : 143	Potassium iodid	1 : 10
Calcium chlorid	1 : 25	Potassium permanganate	1 : 300
Calcium hypochlorite	1 : 1,000	Pure formaldehyd	1 : 25,000
Carbolic acid	1 : 333	Quinin sulphate	1 : 800
Chloral hydrate	1 : 107	Silver nitrate	1 : 12,500
Cupric sulphate	1 : 2,000	Sodium borate	1 : 14
Ferrous sulphate	1 : 200	Sodium chlorid	1 : 6
Formaldehyd (40%)	1 : 10,000	Zinc chlorid	1 : 500
Hydrogen peroxid	1 : 20,000	Zinc sulphate	1 : 20

CONVENIENT FORMULÆ FOR DISINFECTING SOLUTIONS

Bichlorid of Mercury—Corrosive Sublimate.

Bichlorid of mercury	1 dram	1 gram
Water	1 gallon	1 liter

Mix and dissolve. Label *"Poison!"* This is approximately a 1 to 1.000 solution. One ounce of this solution contains very nearly half a grain of corrosive sublimate. Useful for disinfecting clothing, the hands, the surfaces of walls, floors, furniture, etc. Not serviceable for feces or material containing much organic matter.

Formalin.

Formalin	13 ounces	100 c. c.
Water	1 gallon	1 liter

Formalin is a watery solution containing 40 per cent. formaldehyd. The above solution contains approximately 10 per cent. of formalin and is useful for the disinfection of clothing and a great variety of objects. As it has no corrosive action it does not bleach pigments or rot fabrics. When used to disinfect feces twice the above strength should be used.

Milk of Lime.—Slake a quart of freshly burnt lime, in small pieces, with three-fourths of a quart of water, or, more exactly, 60 parts of water by weight with 100 parts of lime. A dry powder of slaked lime (calcium hydroxid) results. Prepare the milk of lime shortly before it is to be used by mixing 1 quart of this dry calcium hydroxid with 4 quarts of water. Air-slaked lime is worthless. Slaked lime may be preserved some time if inclosed in an air-tight container. Milk of lime is especially useful for the disinfection of feces; an equal quantity should be added to the mass and thoroughly mixed.

Carbolic Acid.

Crude carbolic acid (or phenol) ...	7 ounces	50 c. c.
Water	1 gallon	1 liter

The solution is facilitated by dissolving in hot water. This makes approximately a 5 per cent. solution. The addition of from 12 to 14 ounces of common salt to each gallon increases its germicidal power, especially when used for the disinfection of excreta. The crude carbolic acid is more powerful than pure phenol, but can only be used for rough work, such as floors, feces, sputum, etc. For the disinfection of clothing phenol should be used and the solution may be mixed half and half with water, making approximately a 2½ per cent. solution.

Chlorinated Lime ("Chlorid of Lime").

Chlorinated lime	3 ounces	30 grams
Water	1 gallon	1 liter

Mix. This is about a 3 per cent. solution. It is exceedingly powerful and is useful for the disinfection of excreta, privy vaults, cesspools, and many other purposes. It is an active bleaching agent and destroys fabrics in this concentration.

CHAPTER IV

METHODS OF DISINFECTION

A few instances are given upon the following pages of the best methods of disinfecting rooms, excreta, and fomites. The examples selected have been taken as types of a class. In public health work the things most frequently needing disinfection are feces, sputum, and other discharges from the body; bed and body linen, and other fabrics; and bedrooms. The disinfection of water and the pasteurization of milk have already been considered. The disinfection of ships is described under Quarantine.

Air.—It is quite impossible to disinfect the air of a room during its occupancy. In fact, ordinarily little heed need be given to the air itself. Any of the known volatile substances in sufficient concentration to kill microorganisms would render the air unendurable. It is absurd to place such substances as carbolic acid, formalin, or chlorinated lime in an open pan in the sickroom or in the bathroom with the idea that they are serving a useful purpose in disinfecting the atmosphere or in preventing the spread of infection. Occasionally a deodorant, such as formalin, may be used with advantage about the room, but where proper cleanliness and ventilation are observed such substances are rarely called for.

It is of first importance to prevent the infection of the air of the room by taking precautions applicable to the particular infection in question. Thorough ventilation should be maintained, and in this way any chance infection is soon lost by dilution or killed by the sun. An open fireplace is admirable for the ventilation and purification of the air of sick rooms, for by this method the infection is not only carried away, but is destroyed by the heat of the fire in exit. The hanging of sheets wet with bichlorid of mercury or some other germicidal solution at the doorway serves no particular useful purpose.

When a room has become badly infected, say from a case of pulmonary tuberculosis, and there is danger of infection through the dust, it should be given a preliminary fumigation with formaldehyd, which will partly protect the operators who have to take up the carpets or remove the bedding and other articles to the steam sterilizer.

Rooms.—The disinfection of a living-room calls for all the resources of the disinfector's art. The fact that it is necessary to bring the apparatus and materials to the room in order to disinfect it and its contents is one of the main difficulties and will often require the ingenuity and always the vigilance of the operator.

The method to be employed for the disinfection of a room will vary somewhat with the infection for which the disinfection is done. In routine work in the treatment of rooms liable to be infected with a variety of bacterial viruses formaldehyd gas is the most generally useful agent we possess. In the case of yellow fever or malaria insecticides must be selected; in the case of plague our efforts must be directed against rats, mice, fleas, as well as the destruction of the plague bacillus. In cholera and typhoid fever we must pay particular attention to the feces, urine and the objects soiled by them, etc.

Certain articles commonly found in living-rooms, such as bedding, carpets, rugs, cuspidors, upholstered furniture, and other objects liable to become deeply infected must be treated separately by some process applicable to each article. None of the gaseous disinfectants can be trusted to penetrate enough to render articles of this class safe. In case the room is so constructed that it is impracticable to disinfect it with a gas the walls, floors, and all the contents of the room must be disinfected separately in accordance with suitable methods for each case.

Ordinarily carpets and rugs should be left in place until a preliminary gaseous disinfection is accomplished. They may then be taken up and removed for steam sterilization, after which they should be gone over with a vacuum cleaner and finally hung in the sun for a day or two. If carpets, rugs, upholstered furniture, or other articles have become badly contaminated with infected discharges or in other ways the soiled areas should be thoroughly saturated with a strong solution of formalin. Bedding, towels, curtains, clothing, and other articles of like nature may be left in the room exposed to the action of the gas, but should afterwards be removed for boiling, steaming, or immersion in one of the germicidal solutions, as none of the gases can be relied upon for the disinfection of fabrics. Articles removed from the room for disinfection should be placed in a bag or wrapped in a sheet wet with bichlorid of mercury. Rubbish that has collected in the room should be gathered and burned. The cuspidors and their contents require special treatment.

The gaseous disinfectants cannot be depended upon where penetration is required; therefore any article believed to be deeply or badly infected should be treated with another method.

After the room has been properly prepared and all has been made tight, it is filled with the gas according to the method selected. The room should then be sealed in such a way that it cannot be opened without the knowledge of the disinfector. After the proper time has elapsed

the room should be opened by the disinfector himself and the operation should not be considered successful unless there is a distinct odor of the gas present. Windows and doors may then be opened so as to allow the gas to blow away. Cultures of a test organism should always be exposed in order to control the efficiency of the fumigation in each case.

A room which has been carefully treated as above outlined may be considered disinfected, but it is always advisable to follow the disinfecting processes with a very thorough mechanical cleansing and a good sunning and airing.

When a room is to be purified without the use of one of the gaseous disinfectants a somewhat different procedure is followed. Article after article is removed piecemeal and disinfected by an appropriate method. After the room is emptied the walls and their surfaces are flushed, scrubbed, or mopped with bichlorid of mercury, 1 to 1,000, or one of the alkaline cresols.

Stables.—The disinfection of a stable requires a particularly thorough application of all the resources at the hand of the disinfector. The conditions met with in a stable render its disinfection doubly hard, not only on account of the accumulation of organic filth which has worked into the many crevices and saturated the woodwork, but on account of the high resistance of anthrax and tetanus spores, for which stables are sometimes disinfected. In addition to these diseases stables require disinfection on account of tuberculosis, glanders, pleuropneumonia, and various diseases of man as well as those of the domestic animals.

It is advisable to give the stable a preliminary fumigation, preferably with sulphur, in order to destroy surface infection and the vermin which always infest these places. The preliminary disinfection is especially important in the case of plague and glanders, not only to prevent the spread of the infection, but as a safeguard for the disinfectors. Then remove all small articles that need disinfection. The blankets should be wrapped in moist bichlorid sheets and boiled, steamed, or burned. Buckets, currycombs, brushes, stall tools, and other equipment that has been in contact with the sick animals or with infectious materials should be mechanically cleaned with a hot carbolic solution in which they may be allowed to soak over night. Metallic and wooden objects or utensils should be given a thorough preliminary cleansing with a stiff brush and hot water and soap, and then boiled or immersed in a 5 per cent. solution of carbolic acid or 2 per cent. solution of trikresol for several hours. Leather articles, as harness or equipment, should receive a similar preliminary cleansing and be scrubbed with either a strong solution of bichlorid of mercury or carbolic acid.

All hay and grain should be removed from the racks and mangers and all bedding from the floors. After its careful collection at some

designated point this refuse should be saturated with petroleum and destroyed by fire.

The stable must now be soaked with a strong antiseptic solution applied with a hose or splashed on all surfaces by means of mops. The floors, corners, and stalls must be saturated with the solution. On account of the presence of so much albuminous matter carbolic acid or one of its derivatives is preferred for this purpose to sublimate solutions. Now scrape out the débris from all the cracks in the floors and walls; collect it for burning. Then clean the woodwork with hot lye or a strong alkaline soap solution and follow with another general hosing with the antiseptic liquid.

After several days' exposure to air and sunshine the interior of the stable should receive a fresh coat of whitewash, applied quickly, and prepared from freshly burnt lime.

The watering troughs are very apt to be infected, especially in dealing with glanders. In all instances not only the troughs and watering buckets should be disinfected, but the water remaining in them, for often there is no drain or sewer, and this water poured on the ground may be a source of subsequent infection. The water may first be disinfected by the addition of a suitable amount of chlorinated lime or any of the standard germicides. The troughs are then to be mechanically cleaned, thoroughly removing all organic matter, and then applying a strong germicidal solution to both the inside and outside. For metal-lined troughs the use of bichlorid of mercury is, of course, inapplicable, and for such carbolic acid, trikresol, formalin, or potassium permanganate is recommended. Most germicides are poisonous, and must therefore be finally washed out of the trough or buckets by flushing with fresh water and then airing in the sunlight before they are again used. A strong carbolic, formalin, or chlorinated lime solution should be poured down all pipes and drains.

Sometimes the ground in the immediate vicinity of the stable will need attention. Lime will generally be found most useful for this purpose. Carcasses and excreta are to be disinfected and disposed of according to the methods given under these titles.

Railroad Cars.—Railroad cars are rooms on wheels. The principles of their disinfection present nothing novel, but the application presents practical difficulties.

Flat cars or open cars seldom need disinfection, for, even should they become infected, the exposure to the sun and water is sufficient to render them safe from the danger of conveying disease. They may readily be disinfected whenever that may be necessary by scrubbing or flushing them with carbolic acid or bichlorid of mercury solutions.

Freight cars or box cars seldom need disinfection. They sometimes require fumigation on account of mosquitoes, fleas, or rats and mice,

which such cars may carry. Freight cars are best treated for this purpose with sulphur dioxid. In actual practice it will sometimes be found useful to steam them with steam from the locomotive.

Cattle cars and cars used to transport live stock need special attention, particularly if anthrax, tetanus, glanders, foot-and-mouth disease, or tuberculosis is the infection with which they are contaminated. The disinfection of cars of this type is so much like the disinfection of a stable that it is unnecessary to repeat the description here. Cars of this type, as well as all cars, should be kept scrupulously and constantly clean.

Day Coaches and Parlor Cars.—If the disinfection is done as a precautionary measure it is sufficient to fill the coach with formaldehyd gas, which should be followed by a thorough mechanical cleansing. The carpets and rugs and all similar articles, including the upholstered seats and backrests, if removable, should be taken from the car for vacuum treatment and then exposed several hours to the sunshine. The floors should be mopped or scrubbed with one of the germicidal solutions and the spittoons should be well rinsed in a warm carbolic bath and the contents disposed of in one of the ways mentioned under the heading Sputum.

If the disinfection is done on account of known contamination with one of the communicable diseases the car is treated exactly as a room would be under like conditions.

A railroad coach is likely to harbor mosquitoes, flies, and other insect pests that may carry disease; therefore precautions will have to be taken to keep these insects off cars leaving districts infected with yellow fever, typhus fever, malaria, plague, etc., or measures will have to be taken to destroy them after they get on board. As both these requirements are difficult, if not impracticable, it will usually be found best to provide relays at a convenient point and require the passengers to change cars upon leaving an infected for an infectible area.

Sleeping cars present a greater difficulty than any other rolling stock. The berths are apt to become infected and the infective agent may live there a very long time, especially as they are kept closed -almost hermetically sealed, against fresh air and sunshine during the daytime. Much of the difficulty encountered in the disinfection of the sleeping car is due to peculiarities in construction, such as the compact manner in which the bedding is stowed away, the heavy and unnecessary carpets and hangings, the excessive molding and ornamentation of the older type of cars, the use of such materials as plush for upholstering, etc. The wash basins and other objects in the toilet-rooms are liable to contamination with infected discharges from the mouth and nose. The faucets should be so arranged as to permit washing with running water, thus eliminating danger from the bowl.

Before attempting to disinfect the interior of a sleeping car or a passenger coach with one of the disinfectants it is important to close the sashes and all the ventilator openings for the Pintsch gas flames. Much gas will be lost through the open hopper of the water-closet unless that is tamponed. Some cars have a system of ventilating ducts of fresh air entering under the seat or somewhere near the bottom of the car. This must be closed. Formaldehyd gas and hydrocyanic acid gas are practically the only gases which may be used for the treatment of the sleeping car. As these gases lack the power of penetration, all the berths must be opened and all the bedding and other fabrics should be removed for steaming or other treatment. Hydrocyanic acid is especially serviceable for the destruction of bedbugs and vermin which frequently infest sleeping cars.

After the bedding, hangings, carpets, and other fabrics have been removed from the car the toilet-room should be given special attention. The drinking glasses, the wash basins and slabs of the washstands, the brushes and combs, the seat of the water-closet, and other objects liable to infection should be washed or immersed in one of the standard germicidal solutions.

Feces.—The disinfection of feces is most important because these discharges are most dangerous and at the same time most difficult to render safe. Fecal discharges may be disinfected with carbolic acid, cresols, lime, chlorinated lime, or formalin. In hospitals the infected discharges are sometimes boiled in an appropriate vessel with the addition of a deodorizing substance, as potassium permanganate.

From patients the discharges should be received in a glass or impervious vessel containing some of the germicidal substance, more of which is added afterwards, and the mass thoroughly mixed. The mixture should stand at least one hour before the contents are disposed of, and the vessel given a thorough cleansing and disinfection before it is again used. At least an equal quantity of the germicidal solution should be used to the mass disinfected and enough should always be added to entirely submerge the mass. Excreta must always be protected from flies and other insects, even while undergoing disinfection.

Milk of Lime.—Use freshly prepared milk of lime containing 1 part by weight of the freshly slaked lime to 4 parts of water. Add at least an equal quantity to the amount of material to be disinfected and allow the mixture to stand no less than two hours before final disposal. The perfunctory sprinkling of fecal matter with lime or milk of lime, as is often done, is not effective. Lime should not be thrown into the hoppers of water-closets for the disinfection of dejecta, for otherwise a thick mass will accumulate and obstruct the pipes. In disinfecting excreta with lime the reaction of the resulting mixture must be alkaline else the object will not be attained.

Lime or milk of lime is very useful for the disinfection of privies, or trenches in camp, or in country practice. For its use under these circumstances the amount required may be arrived at as follows: The amount of fecal matter per person is reckoned at 400 grams a day. If the urine is also to be disinfected this may be counted as 1,500 to 2,000 c. c. per person daily. For the disinfection of the solid excrement alone 5 grams of lime, or 40 c. c. of the milk of lime (1 to 8), must be reckoned for each person per day. If the urine is included it will take four to five times as much. The mixture must have an alkaline reaction. Attention is again called to the fact that air slaked lime is inert.

Chlorinated Lime.—This is one of the most useful and potent germicidal substances for the disinfection of feces. Use at least a 3 per cent. solution and an amount equal to the mass to be disinfected. Thoroughly mix and allow to stand at least 2 hours. Chlorinated lime combined with air is rendered inert by organic matter; therefore an excess should always be used.

Formalin.—A 10 per cent. solution of formalin may be depended upon to disinfect feces if thoroughly incorporated with the mass and allowed to stand at least one hour. As a deodorant it acts almost instantly.

Carbolic Acid.—A 5 per cent. solution of crude carbolic acid added to an equal bulk of excreta may be depended upon to disinfect in one to two hours, provided the germicide is thoroughly incorporated throughout the mass.

The cresols and the alkaline coal-tar creosotes are valuable agents for the disinfection of fecal matter in small amounts on account of their energetic action and because their efficiency is not greatly impaired by the presence of albuminous matter. As a rule substances in emulsion lack the power of penetration, and if used must be very thoroughly mixed and incorporated with the mass.

Dry earth promotes the disinfection of excreta, thus delaying putrefactive changes while absorbing the odors. It has no inherent germicidal qualities. Corrosive sublimate is unfit for the disinfection of feces and sputum. The discharges from the mouth and nose, not alone of the sick, but of well persons, are often laden with infection. This is one of the frequent means by which disease is transferred. The proper disposal of sputum and its efficient disinfection are therefore an important public health measure to check the spread of tuberculosis, diphtheria, scarlet fever, measles, whooping-cough, influenza, tonsillitis, common colds, pneumonia, the pneumonic form of plague, etc. It is a good rule to require the discharges from the mouth and nose of all hospital patients to be received upon small pieces of gauze or in individual cups which may subsequently be burned.

Sputum.—The most trustworthy chemical disinfectants for sputum

are carbolic acid, 5 per cent; formalin, 10 per cent. or stronger; chlorinated lime, 3 per cent.

Sputum should be kept well covered in suitable receptacles until it is disposed of. Simply keeping water in the bedside cups or in cuspidors will prevent whatever slight danger exists in the dissemination of infection from such sources. Antiseptic solutions may be used for this purpose, but are not necessary.

The disinfection of the large amounts of sputum such as that collected in hospitals, public buildings, and other places is a difficult and disagreeable task. On account of its dense consistency it prevents the penetration of chemical solutions. A very good apparatus for the disinfection and disposal of sputum in hospitals, sanatoria, etc., consists of an autoclave in which the material is steamed under pressure and at a temperature of 120° C.; after the completion of the process the disinfected mass is washed through the drain into the sewer by water entering the autoclave. The entire operation can thus be conducted under cover. Dr. Wm. J. Manning[1] describes an ingenious and efficient method of handling spittoons and disposing of the sputum at the Government Printing Office in Washington. The cuspidors are self-draining. They are collected and handled by devices so that the attendants do not have to handle them directly.

Bed and Body Linen.—Fabrics, such as towels, napkins, handkerchiefs, sheets, pillowslips, and similar articles, should always be disinfected after contact with any of the communicable diseases, for they are very apt to become infected. They may be steamed or boiled or immersed in a germicidal solution such as carbolic acid, 5 per cent.; formalin, 10 per cent.; or bichlorid of mercury, 1 to 1,000.

Special care is necessary in washing or disinfecting towels, sheets, underwear, and other fabrics soiled with such discharges as pus, blood, or excreta. If they are heated or boiled without special precautions they will become indelibly stained by the coagulation of the albuminous matter which becomes fixed in the fiber.

Soiled wash may be treated as follows: It is wrapped in a sheet wet with sublimate solution, and this placed in a sack likewise moistened with a germicidal liquid. The sack is placed unopened in a solution containing 3 per cent. of soft soap and heated to 50° C. for three hours and left in the same solution forty-eight hours after it cools. If not soiled with albuminous matter the wash may be immersed in a solution of bichlorid of mercury 1 to 1,000, with the addition of common salt. After this preliminary disinfection the articles are boiled half an hour in a water containing:

[1] *J. A. M. A.*, Sept. 11, 1909, Vol. LII, pp. 829-832.

Petroleum	10 grams
Soft soap	250 "
Water	30 liters

Books.—With the exception of their external surface, books cannot be disinfected in the bookcase or on the shelves of houses and libraries. However, if the books have not been handled or exposed to infection in any way except by their presence in the sickroom there is no reason for considering any part of the book, except the exposed surface, as infected. Such books may be disinfected by exposing them to formaldehyd gas without first disturbing them in any way.

Books which have been handled by the patient or which have been otherwise exposed to infection require particular care in their disinfection on account of the difficulty of penetrating between the leaves. Books used in public libraries are often regarded with suspicion, and many librarians require that they should be sunned, aired, or disinfected before they are again issued. The danger from this source has doubtless been exaggerated. Books, however, which have been handled by persons suffering with one of the readily communicable diseases should always be disinfected before they are again used.

Books may be disinfected in a specially constructed chamber by means of heat and formaldehyd gas. They must be arranged to stand as widely open as possible upon perforated wire trays. Under these conditions the exposure should be continued twelve hours with high percentages of formaldehyd and a temperature of 80° C., a partial vacuum having first been introduced. The binding, illustrations, and print of books are not injured by this process.

When only a few books are to be treated in the absence of a special apparatus they may be disinfected by placing 2 or 3 drops of a 40 per cent. formalin solution on every second page, taking care to distribute the drops well. The book is then laid in a close box or drawer in which more formalin has been sprinkled, and left in a warm place for not less than twenty-four hours.

Pamphlets and unbound volumes may be steamed without serious harm. Steam is not applicable to the disinfection of bound books on account of the glue and leather.

Beebe[1] recommends dipping the books in a solution of carbolic acid and gasoline. After immersion the books should be placed before an electric fan, which rapidly drives off the gasoline.

Nice[2] recommends the use of moist, hot air at 80° C. and 30 or 40 per cent. humidity for thirty-two hours for the disinfection of books. This is said to destroy all non-spore-bearing bacteria in closed books,

[1] *Jour. Am. Public Health Assn.*, Vol. I, No. 1, p. 54, Jan., 1911.
[2] *J. A. M. A.*, April 20, 1912, Vol. LVIII, No. 16, p. 1201.

even tubercle bacilli in thick layers, without injuring the most delicate bindings.

Cadavers.—Dead bodies may be the cause of the spreading of some of the communicable diseases. The body without previous washing should be wrapped in a sheet wet with a strong germicidal solution, such as bichlorid of mercury, 1 to 500; carbolic acid, 5 per cent., or trikresol, 1 per cent., until it is disposed of. Should it be desirable to wash the body it should be done with formalin (10 per cent.) or Labarraque's solution, or one of the germicidal solutions above mentioned.

From a sanitary standpoint bodies dead of one of the communicable diseases are best disposed of by burning. When cremation is not practicable the body may be surrounded by twice its weight of freshly burnt lime in an hermetically sealed coffin and buried at least 6 feet underground. There is much less danger from the spread of disease from bodies buried in the ordinary way than is commonly supposed.

Embalming with strong solutions of formalin and arsenic that are commonly used for this purpose is effective in destroying all but the surface infection.

The disposal of bodies dead of anthrax is an important and difficult matter and has been discussed on page 285.

Thermometers.—A thermometer may be the source of conveying disease from one person to another, and it behooves the physician to exercise special care concerning its cleanliness and disinfection. The best practice is to keep pure formalin in the thermometer case in which the instrument is kept constantly bathed.

Wells and Cisterns.—The disinfection of a well may be accomplished by the use of freshly burnt lime. About half a barrel is thrown into the well, stirred up with the water, and the walls are scrubbed down with the resulting milk of lime. The well is then pumped out, cleaned, allowed to refill, and a second supply of lime added, after which the well is allowed to stand twenty-four hours. After a thorough stirring the solution is then pumped out and the well is allowed to refill and is reemptied until the water is practically free from lime. Instead of lime chlorinated lime may be used for this purpose, sufficient being added to make approximately a 1 per cent. solution.

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